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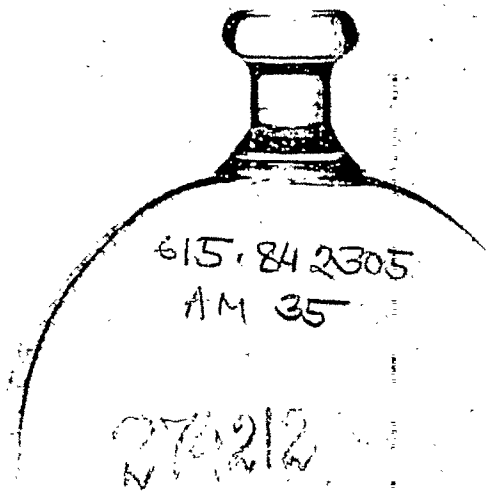
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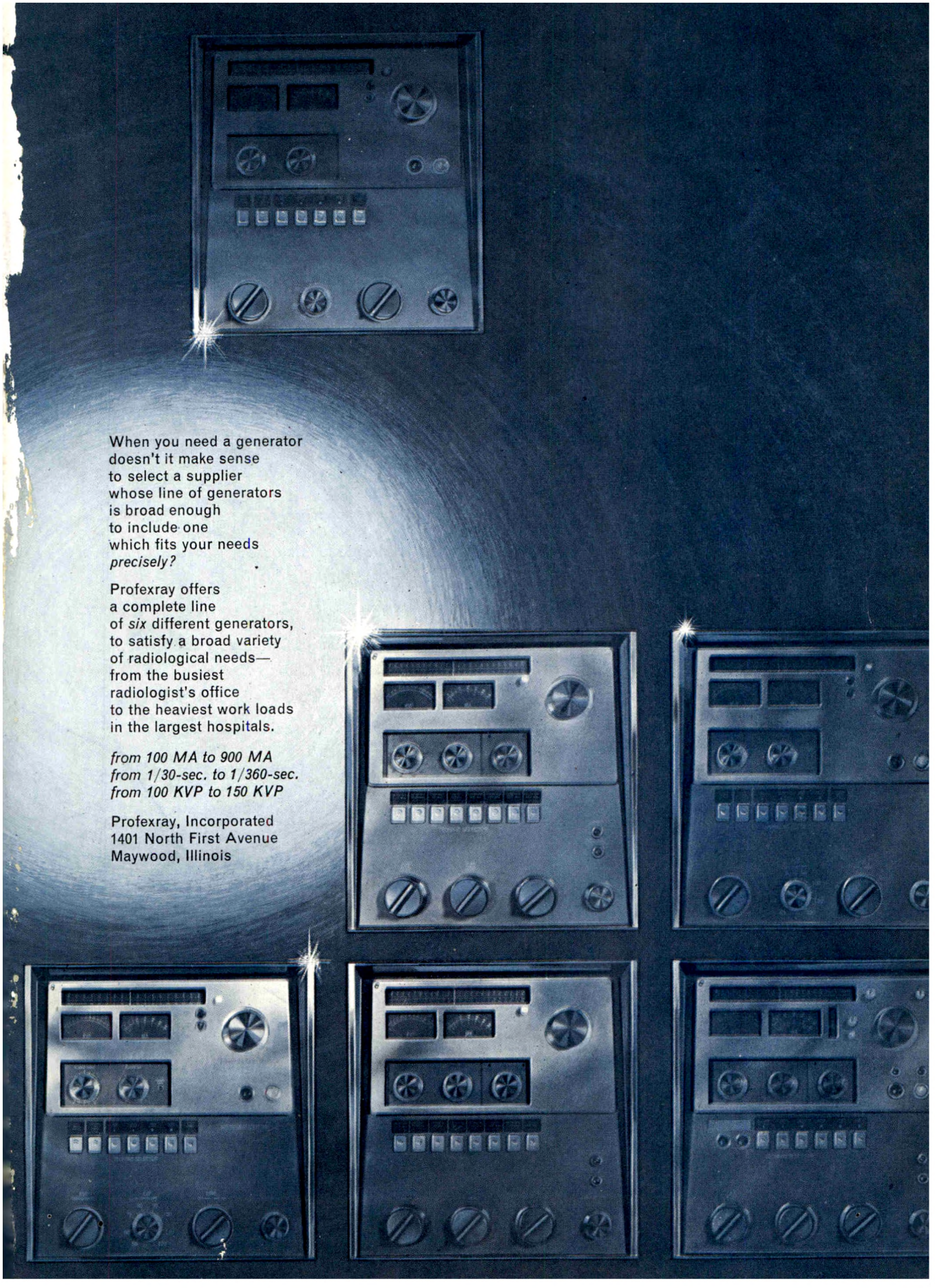
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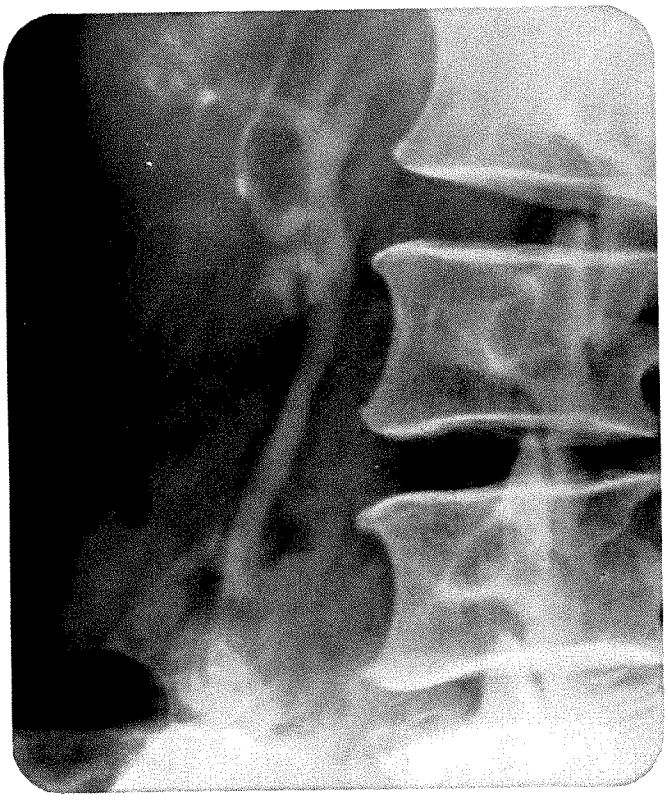
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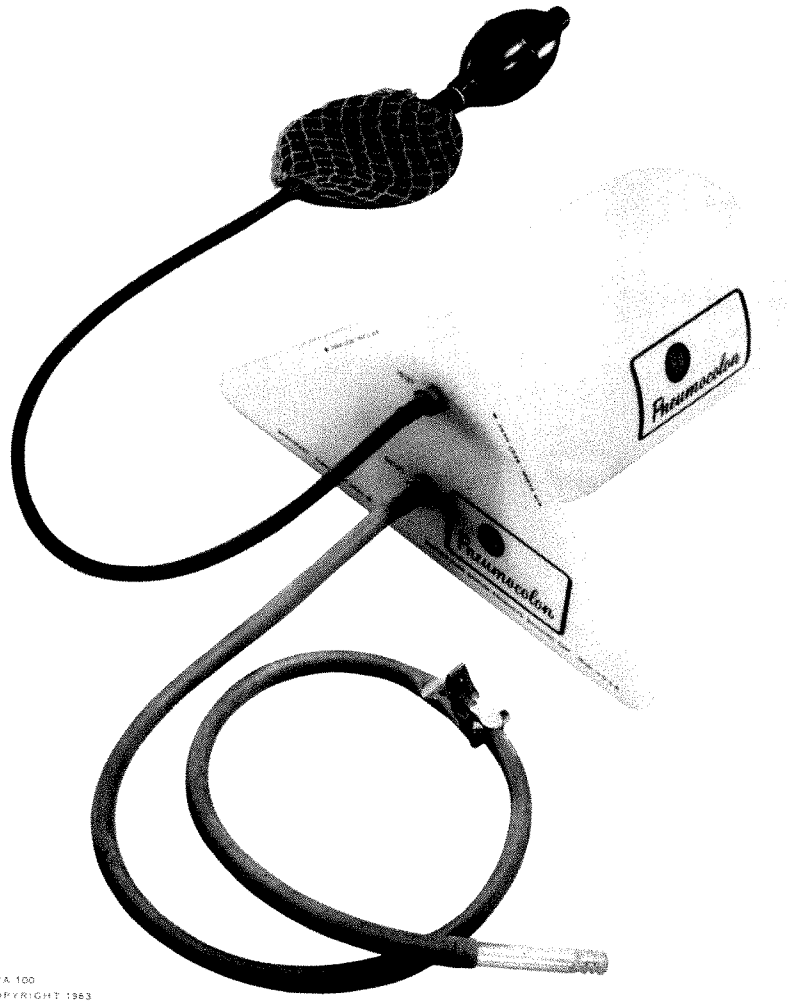
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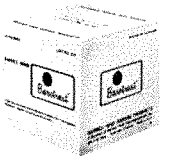
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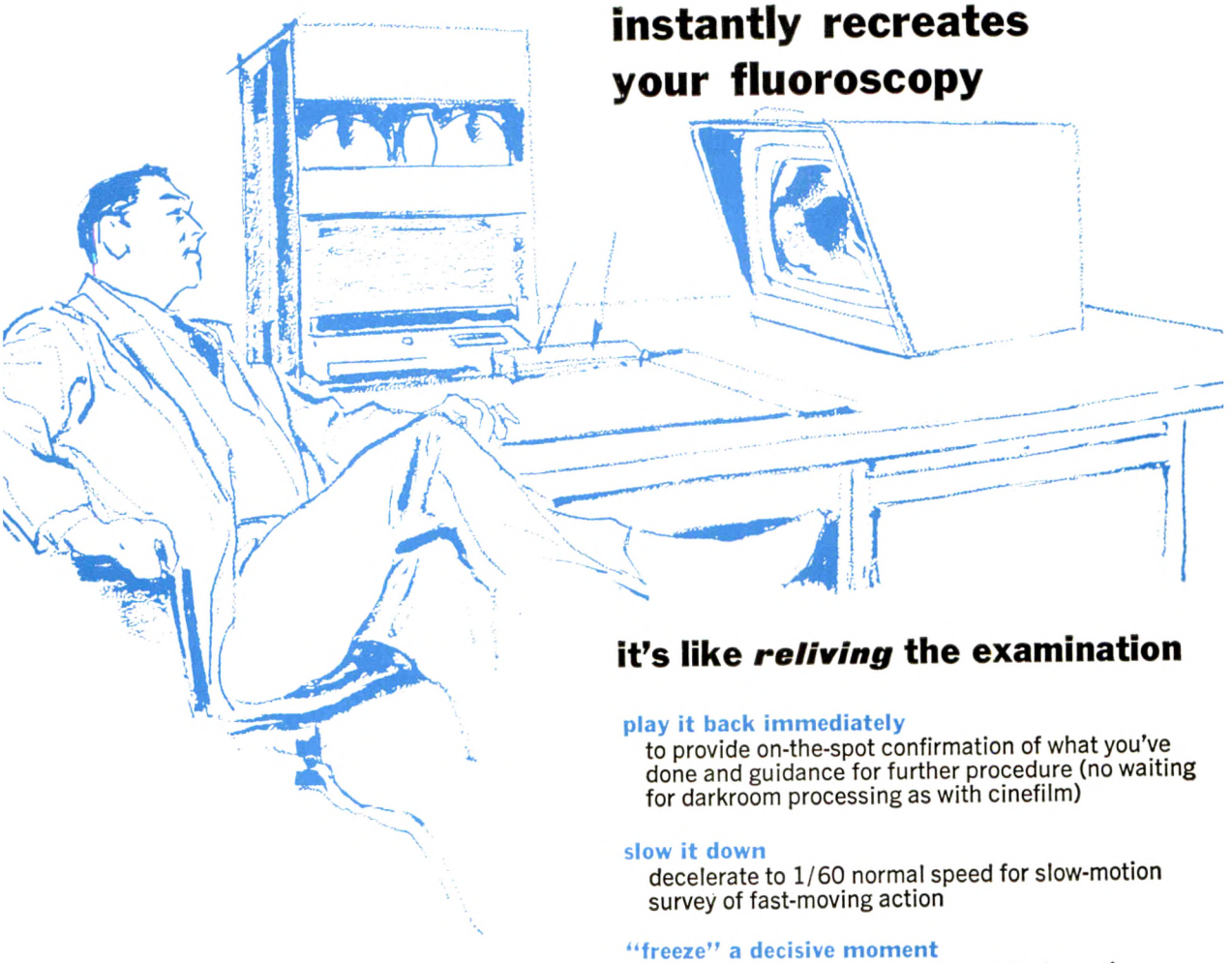
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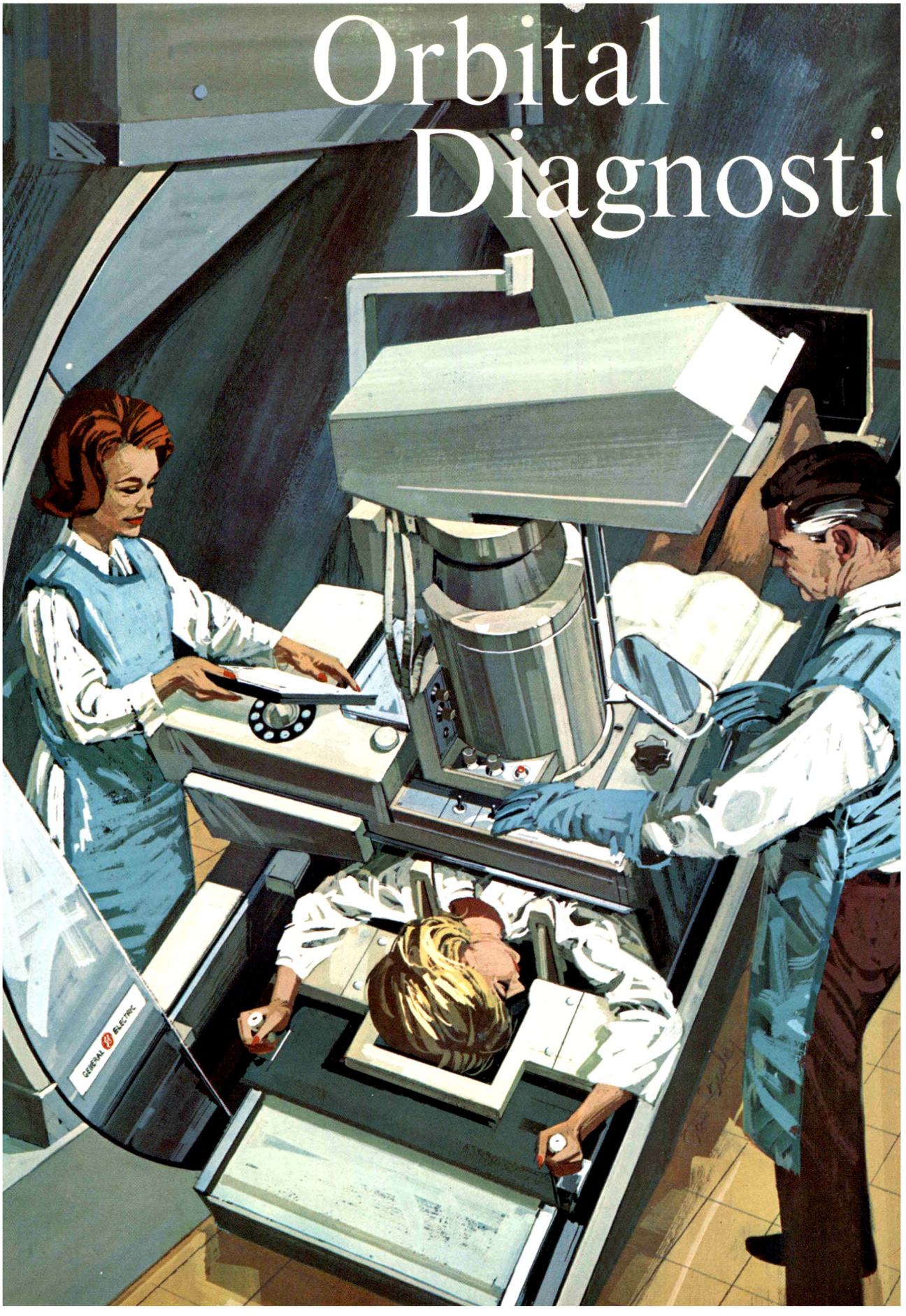
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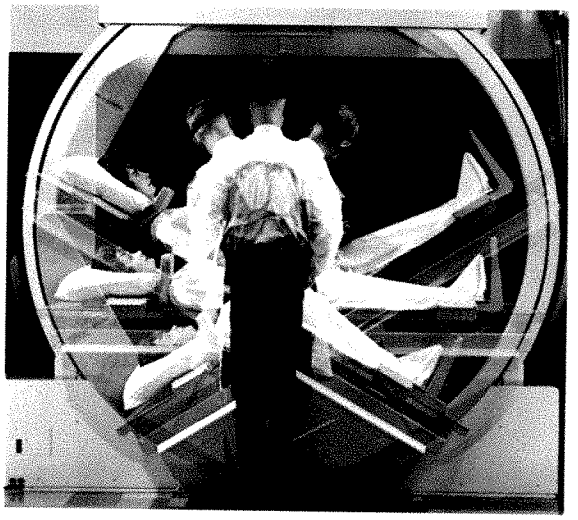
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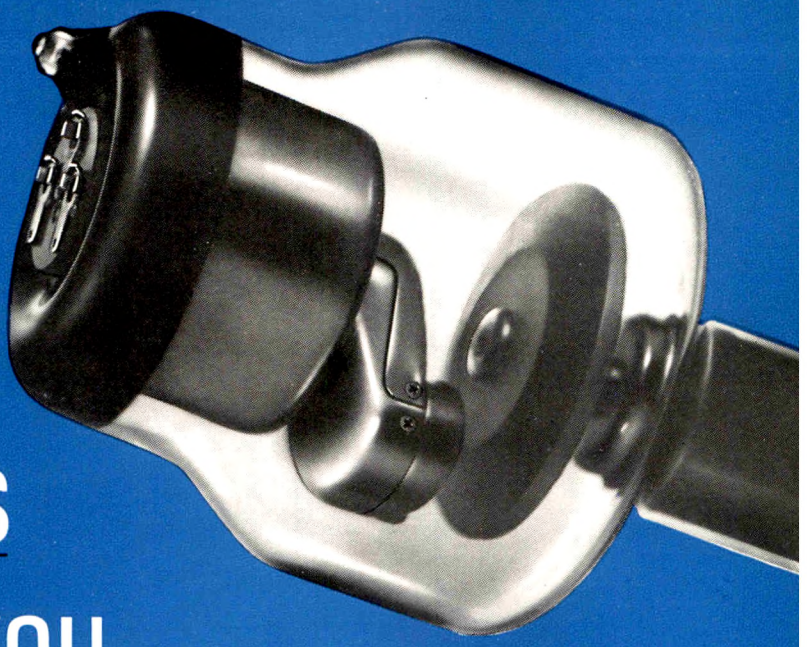
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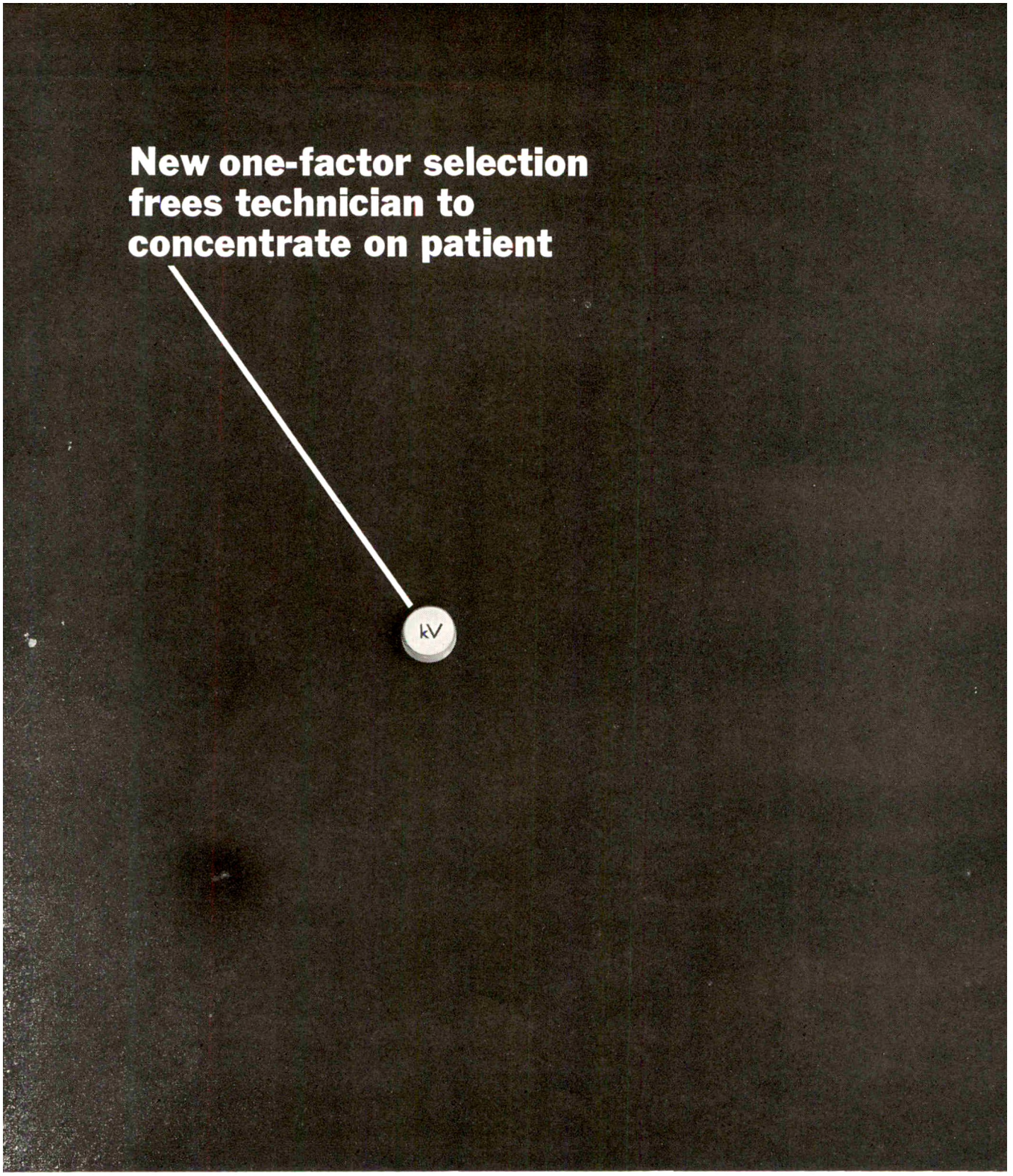


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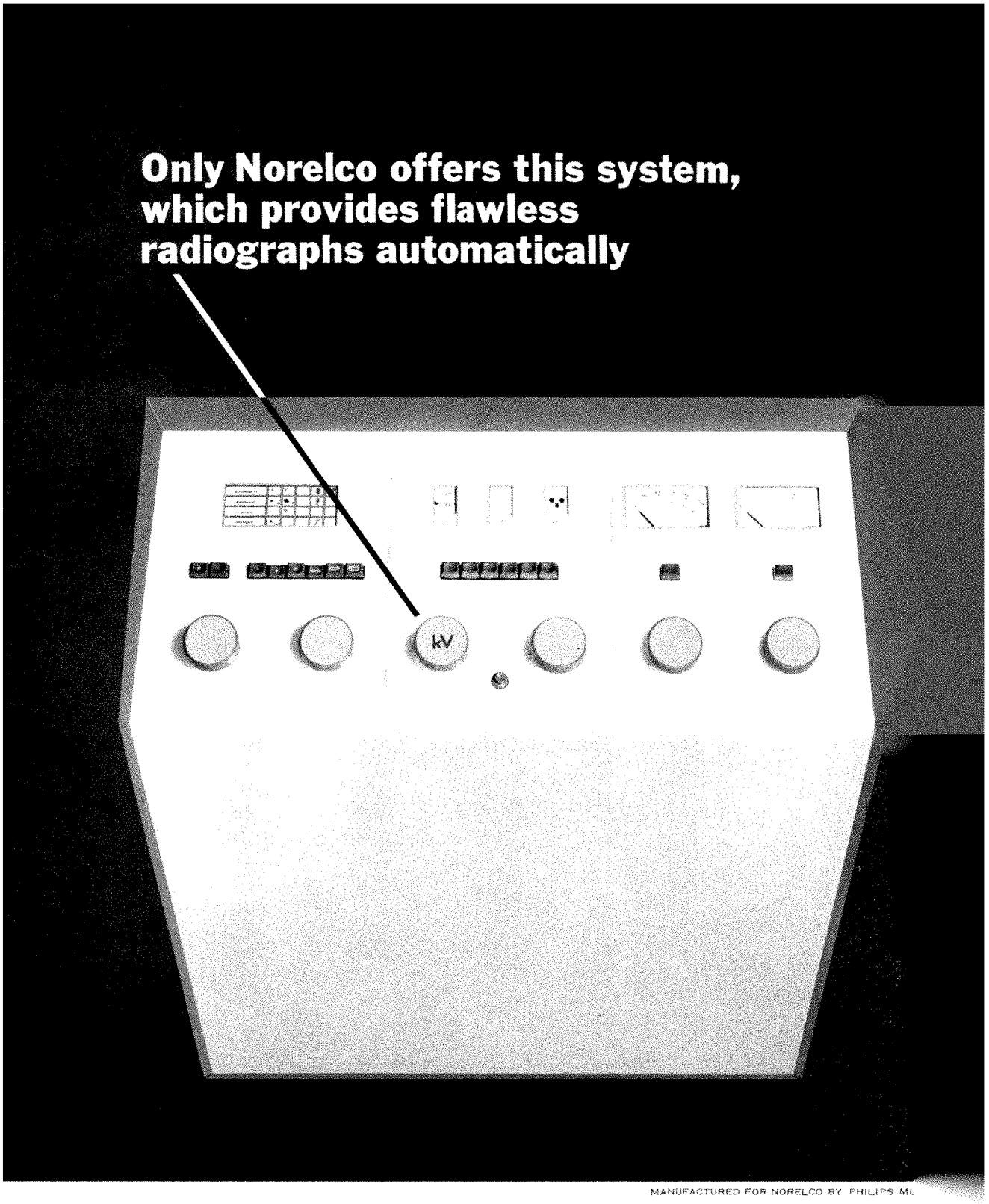


kV is the only factor your technician is required to select. Once that is done, she presses the exposure button and the automatic system does the rest.

Proper density is guaranteed by the Amplimat, the automatic ionization-type exposure control which measures the average X-ray quantum acting on the film over several selectable areas. To ensure positive positioning, the lightbeam indicator projects the location of the measuring areas on the patient. Just as in the case of the modern camera, automatic exposure with Amplimat pays off in consistent production of optimum radiographs.

Predictable contrast is guaranteed by

Only Norelco offers this system, which provides flawless radiographs automatically



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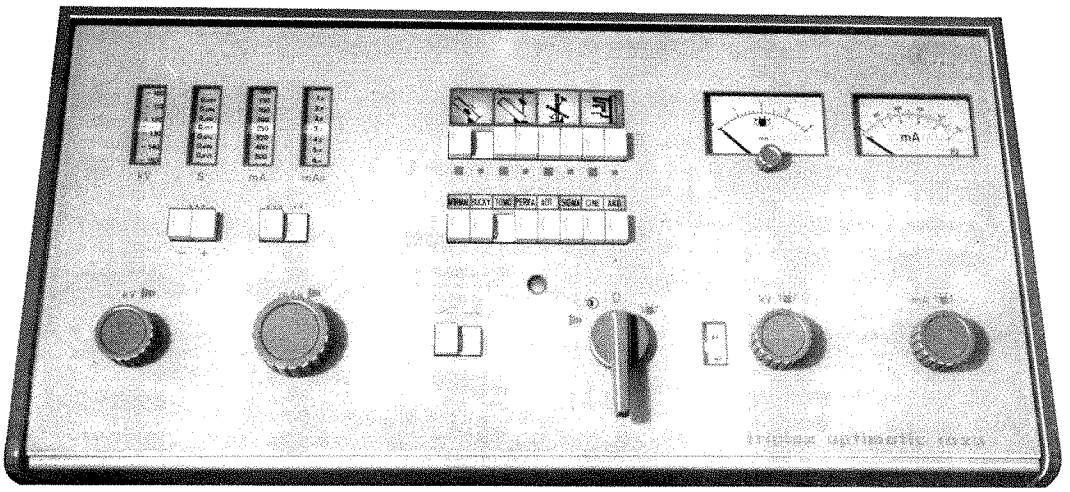
automatic line correction and by the essentially constant voltage output characteristic of a 3-phase generator.

Sharpness is increased by the 'continuously falling tube-load principle', which permits shortest possible exposure time with maximum available tube loading. Optimum sharpness is assured when taking advantage of the high load characteristic and small focal spot sizes of the Super Rotalix tube.

Our systems engineers are available, at your convenience, to discuss this system.

North American Philips Company, Inc., *Medical Division*, 100 East 42nd Street, New York, New York 10017. In the Chicago area: 749 Howard Street, Evanston, Illinois.

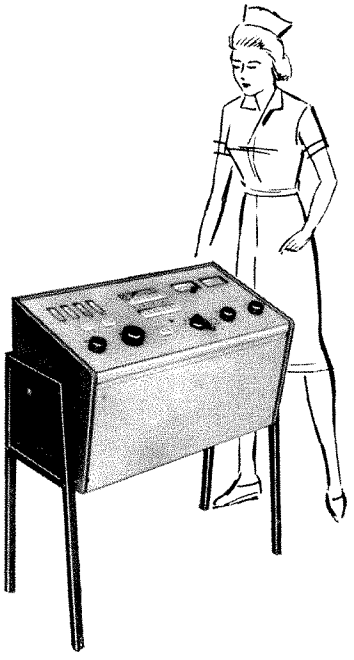
Norelco



Elema-Schönander three-phase x-ray equipment

Triplex Optimatic

with . . .



- Mechanical or electronic contactor
- High tension transformer equipped with selenium rectifiers in twelve-pulse system
- Operation of up to four double-focus X-Ray tubes
- Capacity—1000mA at 100kV
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- Automatic tube protection
- Exposure rates:
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  Mechanical contactor—to 8/sec.

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...in gold, enamel, pearls, the Rospigliosi Cup, exquisite masterwork of Benvenuto Cellini, ranks with the best decorative pieces of the Renaissance. Today, research development and dedication make Gevaert medical X-ray film one of the best you can buy...or use!

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Five Radiation Lab Functions You Can Perform Best with a GAMMASCOPE®



1. Using mixed tracers in absorption studies

The 100-channel Gammascope will function as a dual-peak spectrometer, clearly displaying the energy peaks of both elements on the visual and printed spectrum. Both elements are counted automatically and simultaneously.

2. Working with short-lived isotopes

Half-lives of less than a day are problems for scanning devices, but the Gammascope, with fast automatic data accumulation, can complete a spectrum analysis in far less time than it takes to materially affect the isotope's energy.

3. Determining isotope purity

Monitoring samples to determine their purity or to check the specifications of matched samples are other laboratory processes that can be completed quickly and accurately with the greater resolution, counting speed and readout efficiency of the Gammascope.

4. Using several isotopes in succession

It is a simple matter to recalibrate the Gammascope for each new element used. Simply set the adjustable visual window to intensify the primary energy peak. The window adjusts to any width (number of channels) and any location on the energy spectrum.

5. Making diagnostic and experimental spectrum analyses

In whole body counts, uptake studies and other biophysical radiation applications, the Gammascope will complete a spectrum analysis in a fraction of the time a scanning spectrometer takes. All pulses are stored in the 100-channel magnetic core memory while the cathode-ray tube simultaneously displays the build-up of the spectrum. To make a complete analysis you calibrate in one step, start the analysis and the automatic accumulation takes over. The completed count — determined by the pre-set live timer — can be printed out on the digital printer.

The Gammascope pulse analysis system includes built-in linear amplifier, high voltage supply, visual display and external printer — $5990 (export slightly higher).

For complete data contact the nearest TMC office or Technical Measurement Corporation, 441-3 Washington Avenue, North Haven, Connecticut.

TMC

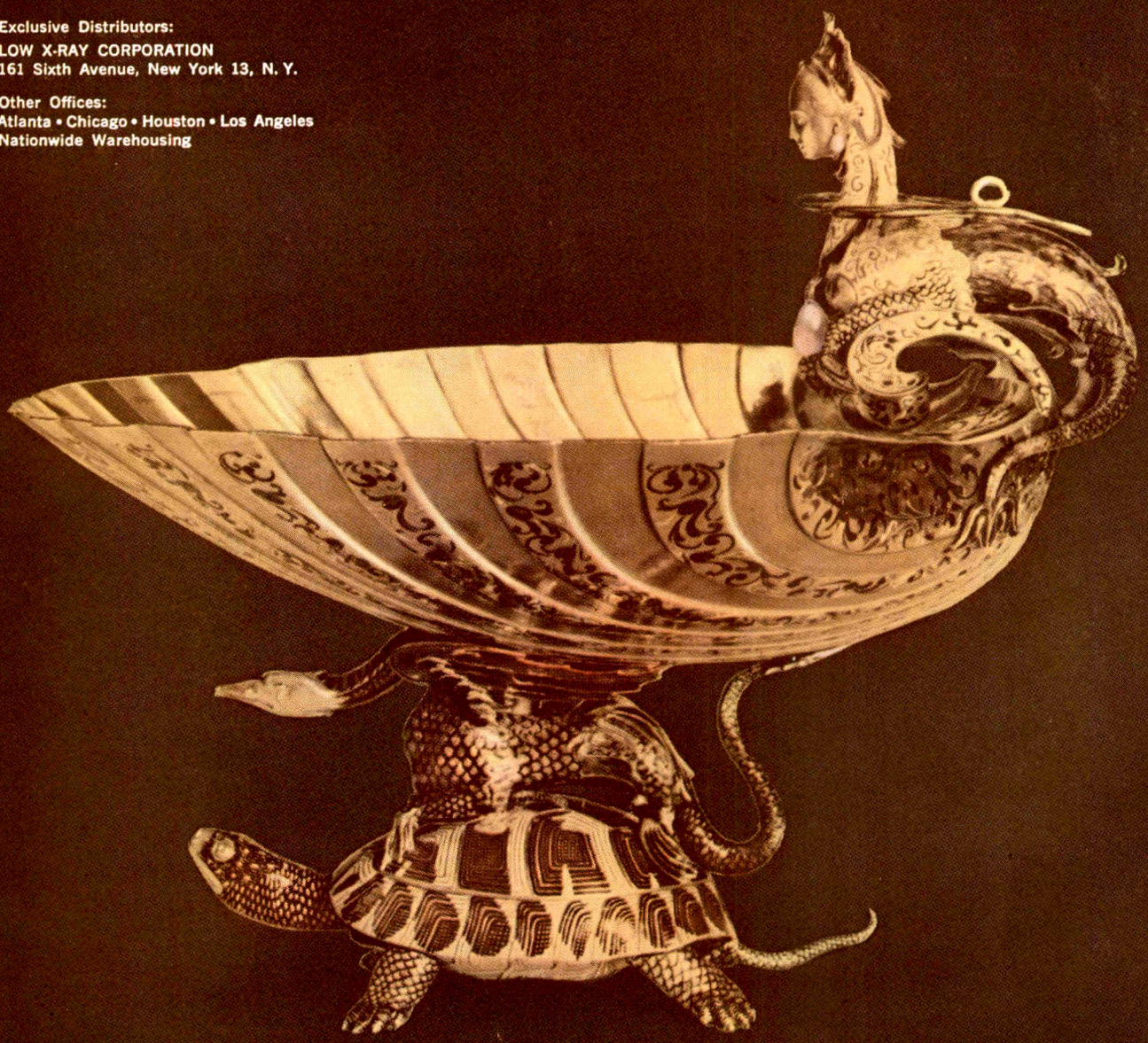
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...in gold, enamel, pearls, the Rospigliosi Cup, exquisite masterwork of Benvenuto Cellini, ranks with the best decorative pieces of the Renaissance. Today, research development and dedication make Gevaert medical X-ray film one of the best you can buy...or use!

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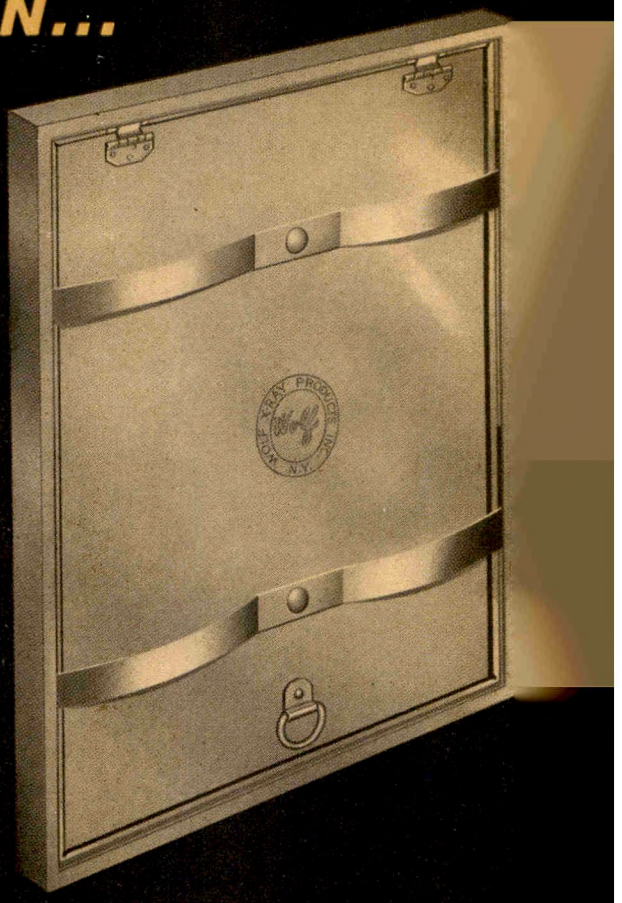
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For complete data contact the nearest TMC office or Technical Measurement Corporation, 441-3 Washington Avenue, North Haven, Connecticut.

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TAKING THE NATION...



...by storm!

Perfect contact, sharp definition, pinpoint elicitation of detail, and ease of handling characterize the quality-concept of Wolf cassette construction. The perfection of Wolf craftsmanship is your assurance of the ultimate in film security for all the applications of cassette utilization.

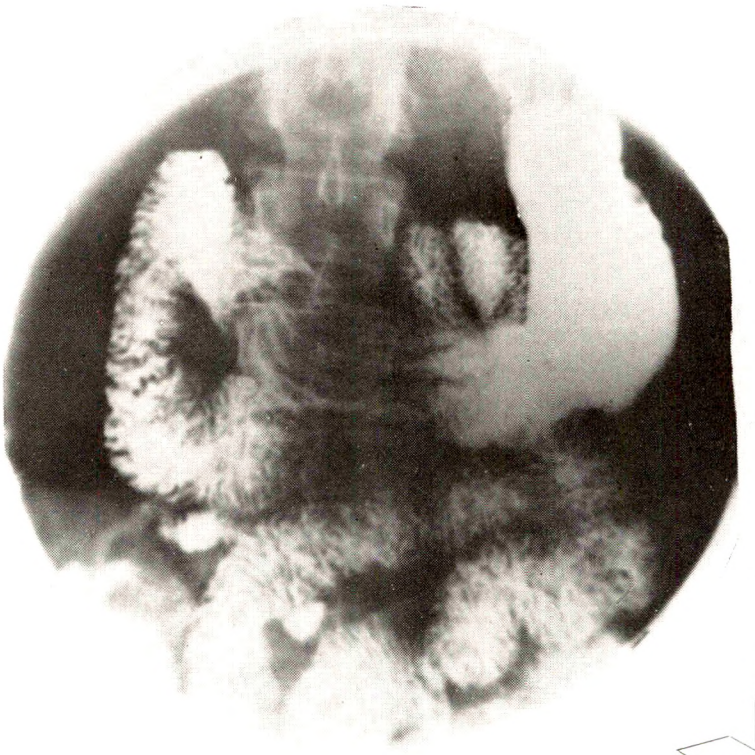
Wolf cassettes are available in both economy and deluxe styles, in *all* sizes in use today (including centimeter sizes) and may be had with photo timing as well. And here's good news! . . . When ordering a Wolf cassette be sure to ask for Wolf intensifying screens to be included . . . It will be our pleasure to mount them for you!

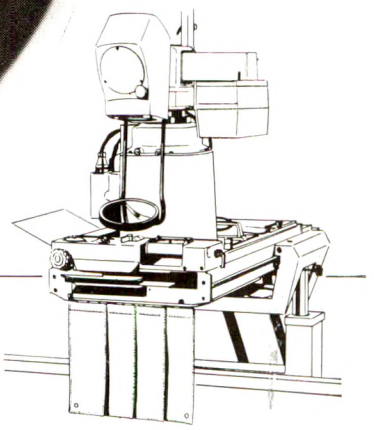
For a comprehensive evaluation of the Wolf cassette, consult with your dealer. He will be *most* pleased to serve your interest . . . and remember quality is a matter-of-fact condition at Wolf—where we are dedicated to Creating Tomorrow's Traditions . . . Today.

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LARGE-SIZE INFORMATION ON 70 MM FILM

The 70 mm spot-film camera faithfully records the highly informative image produced by the 9" Image Intensifier. Definition approaches that of normal full-size radiographs.
Application of this camera means that: The X-ray dose can be reduced by a factor ten to twenty per exposure • Considerable savings can be effected in film material • Less filing space is required • The 0.3 mm focus can be used for radiography. *The camera is tuned to efficiency in the broadest sense.* • The amount of film in the feed cassette can be checked • The feed cassette has a capacity of 30 metres of normal 70 mm film; the take-up cassette holds maximum 3 metres: equal to 40 exposures • A built-in cutter provides for immediate removal of single lengths of film down to one frame • Automatic threading of the film onto the spool eliminates loss • A film marker numbers each exposure photographically to simplify identification and filing • An exposure counter synchronized with the marker, indicates the relevant number.

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"Got
the
processor
unjammed,
Miss Jones?"

"I'm
Miss Smith–
and I wish
you'd get
CRONAR
film so this
machine
wouldn't jam
so often"

"Machine working better with CRONAR, Miss Smith?"

"I'm Miss Jones– you've never seen me with the lights on"



You'll see many pleasant changes when you get a film with CRONAR* polyester film base. CRONAR is almost impervious to water and processing solutions. That's why CRONAR films don't go limp in automatic processing units—these films transport better, with less chance of jamming.

Three major hospitals, where processors jammed several times a month (or a week!), switched to CRONAR—and haven't had a shutdown in a year and a half. Ask your Du Pont Technical Representative or your Du Pont Dealer about other advantages of CRONAR base. Or write us at: Wilmington, Delaware 19898. After all, isn't it worth an effort to see Miss Jones in the daylight?

*Du Pont's registered trademark for its polyester film base

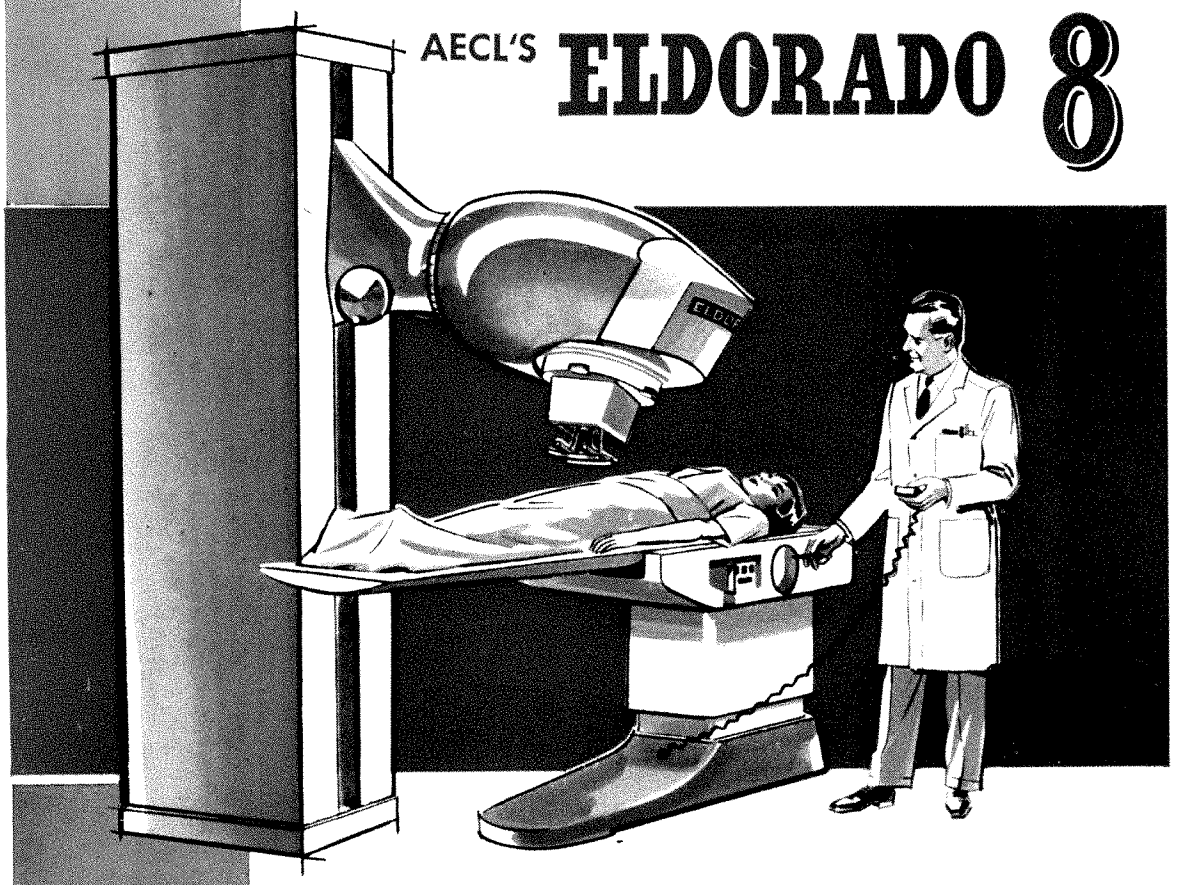
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The CRITERION of performance and economy

AECL'S ELDORADO 8



Designed for precision and convenience in fixed beam and multiple port techniques — at very low cost of purchase, operation and maintenance.

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- **comprehensive beam control**
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- **wide choice of accessories.**

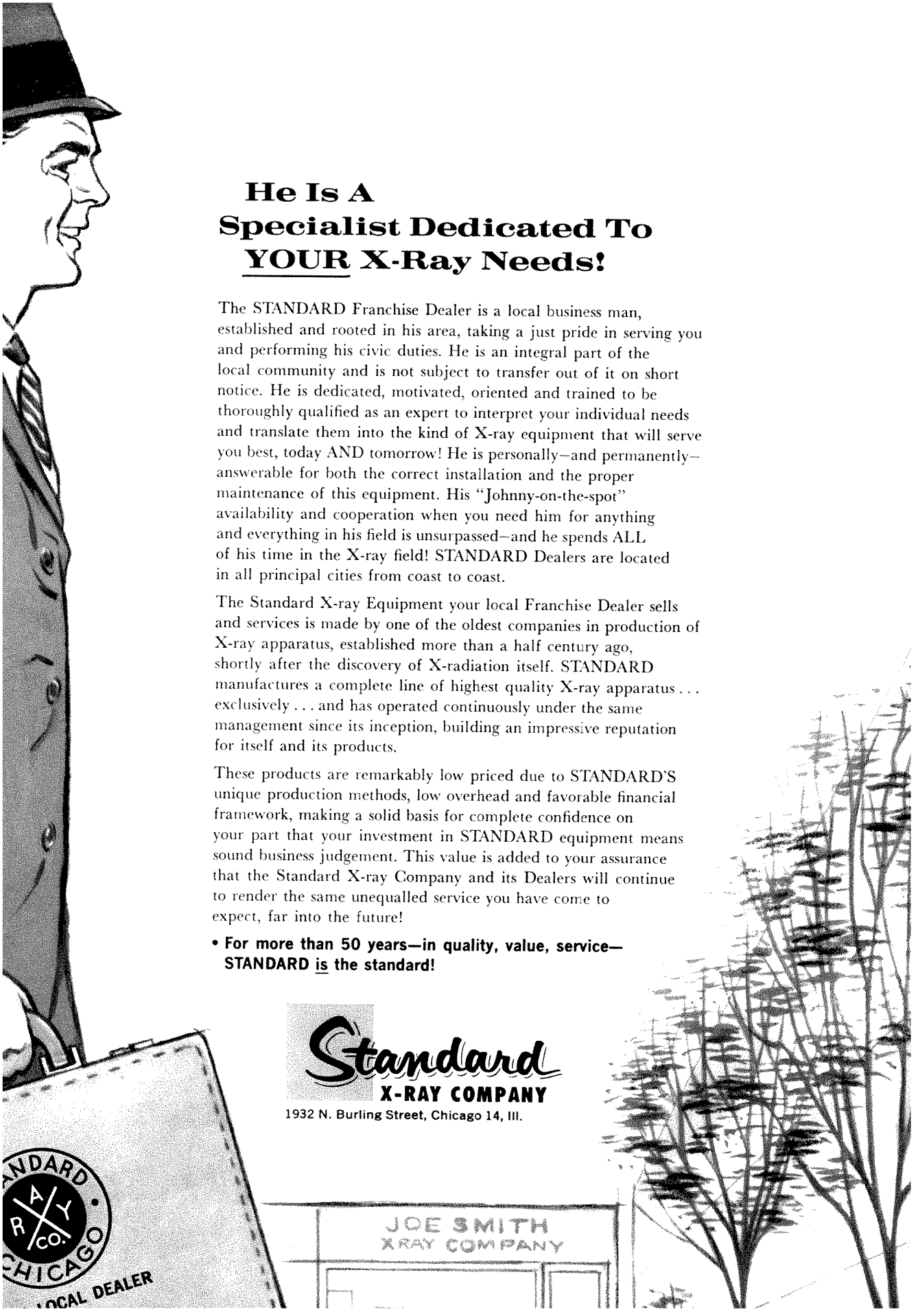
The ELDORADO 8 is one of A.E.C.L.'s new generation of cobalt units. Based on 13 years of experience, these units have been entirely designed since 1960 and meet the sophisticated requirements of the up to date clinic.

For full information on the Eldorado 8 or A.E.C.L.'s other new units, please write:

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The STANDARD Franchise Dealer is a local business man, established and rooted in his area, taking a just pride in serving you and performing his civic duties. He is an integral part of the local community and is not subject to transfer out of it on short notice. He is dedicated, motivated, oriented and trained to be thoroughly qualified as an expert to interpret your individual needs and translate them into the kind of X-ray equipment that will serve you best, today AND tomorrow! He is personally—and permanently—answerable for both the correct installation and the proper maintenance of this equipment. His "Johnny-on-the-spot" availability and cooperation when you need him for anything and everything in his field is unsurpassed—and he spends ALL of his time in the X-ray field! STANDARD Dealers are located in all principal cities from coast to coast.

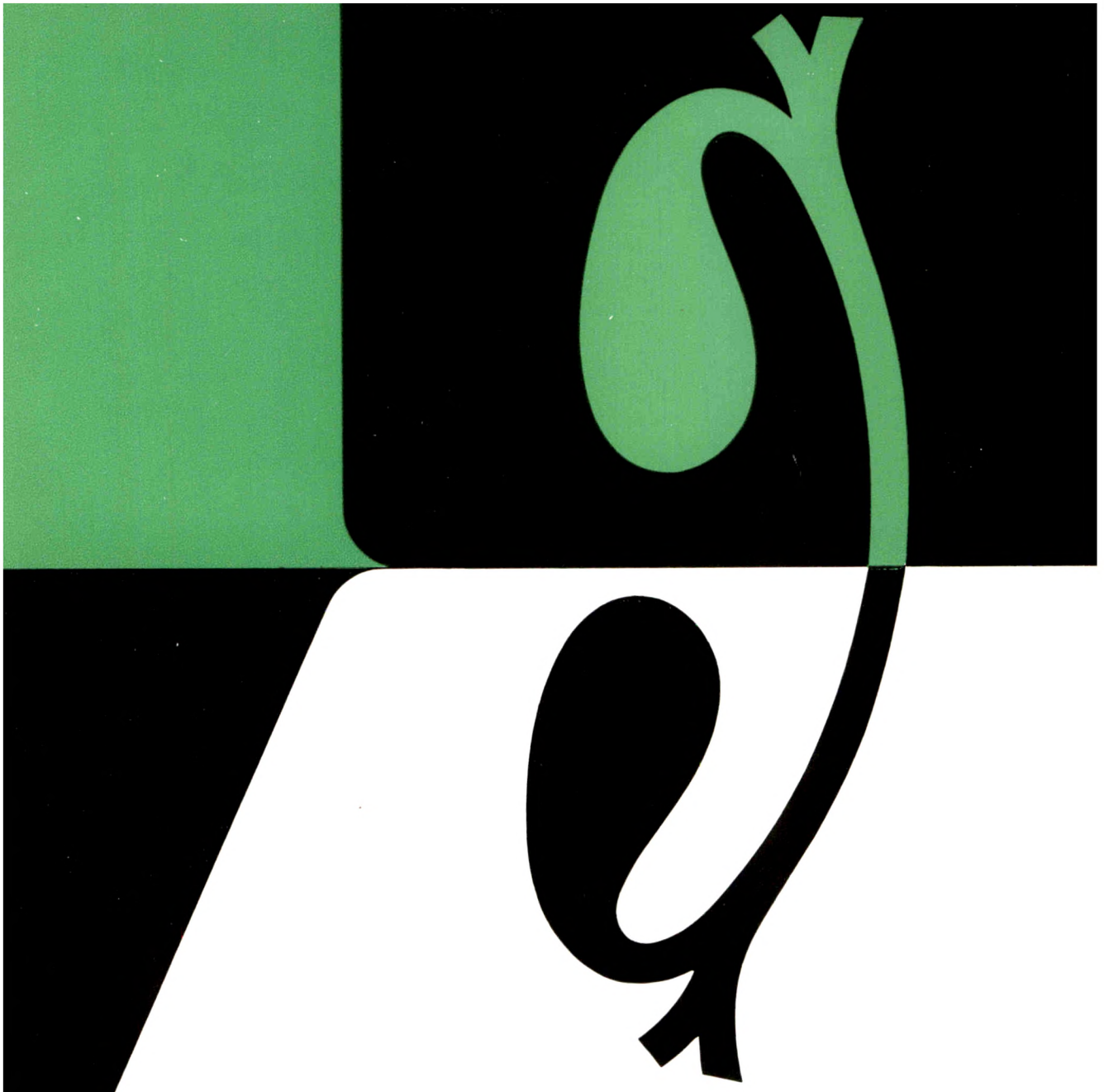
The Standard X-ray Equipment your local Franchise Dealer sells and services is made by one of the oldest companies in production of X-ray apparatus, established more than a half century ago, shortly after the discovery of X-radiation itself. STANDARD manufactures a complete line of highest quality X-ray apparatus . . . exclusively . . . and has operated continuously under the same management since its inception, building an impressive reputation for itself and its products.

These products are remarkably low priced due to STANDARD'S unique production methods, low overhead and favorable financial framework, making a solid basis for complete confidence on your part that your investment in STANDARD equipment means sound business judgement. This value is added to your assurance that the Standard X-ray Company and its Dealers will continue to render the same unequalled service you have come to expect, far into the future!

- **For more than 50 years—in quality, value, service—STANDARD is the standard!**

Standard X-RAY COMPANY

1932 N. Burling Street, Chicago 14, Ill.



In cholecystography coming events will cast their shadows

The need for gallbladder surgery, for example. *Better contrast visualization*, of course means more *precise diagnosis* — the reason why well-tolerated Telepaque (brand of iopanoic acid) has so firmly established itself in oral cholecystography and cholangiography. Telepaque not only provides gallbladder shadows with better contrast, and improved visualization of the ducts, but it does so without disturbing gallbladder physiology. Convenient and economical, too.

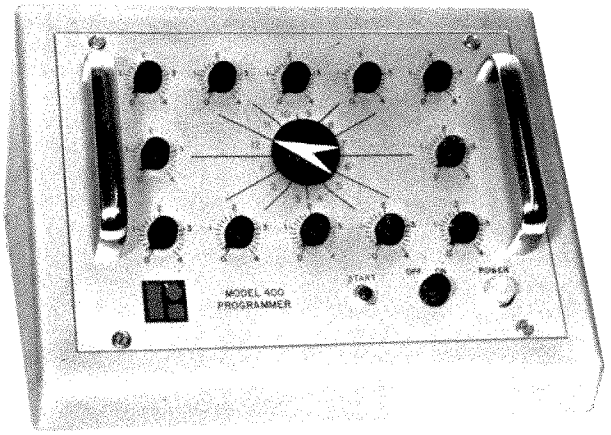
Side effects, which are usually mild, include nausea, vomiting, diarrhea, dysuria. *Contraindications* are acute nephritis and uremia, and gastrointestinal disorders which prevent absorption of the medium.

Usual Dosage: 3 Gm. (6 tablets) at night after a light supper. Tablets of 500 mg., envelopes of 6 tablets, boxes of 5 and 25 envelopes; and bottles of 500.

well-tolerated medium with distinctly better contrast **Telepaque®**

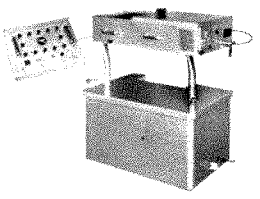
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Please address your inquiries to: The Automatic Seriograph Corp., 4900 Calvert Rd., College Park, Maryland. Our telephone number is: UNion 4-5678.

THE AUTOMATIC SERIOGRAPH CORPORATION
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Dependability



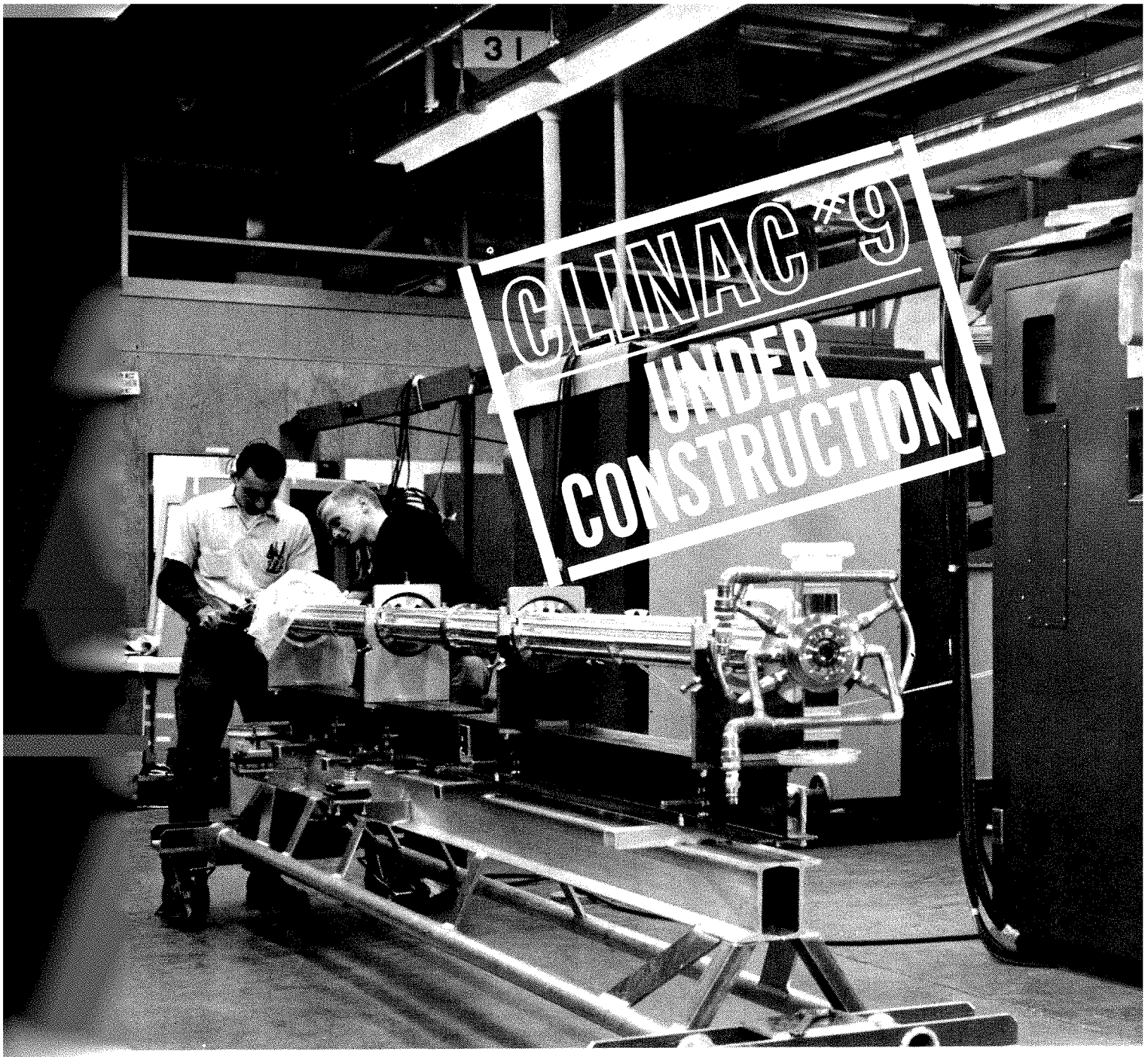
In the key decisions of diagnosis, you must rely professionally on radiographs in which you can place full confidence. *Speed* that reduces risk of involuntary movement on the part of the subject, that sharpens detail; *gradation* that unfailingly produces a wide range of opacities with correct contrast; these properties are vital to consistent quality and to avoid wasteful rescheduling of procedures. Other important factors are fineness of grain and the utmost in reproducibility. All are characteristic of Ilford Red Seal X-ray Film, crafted in the finest British tradition. Each package bearing the Ilford Red Seal label is the product of continuing research and highest scientific standards. Red Seal X-ray Film in all standard sizes and bulk pack, together with Ilford Chemicals and Intensifying Screens, are available through your regular dealer.

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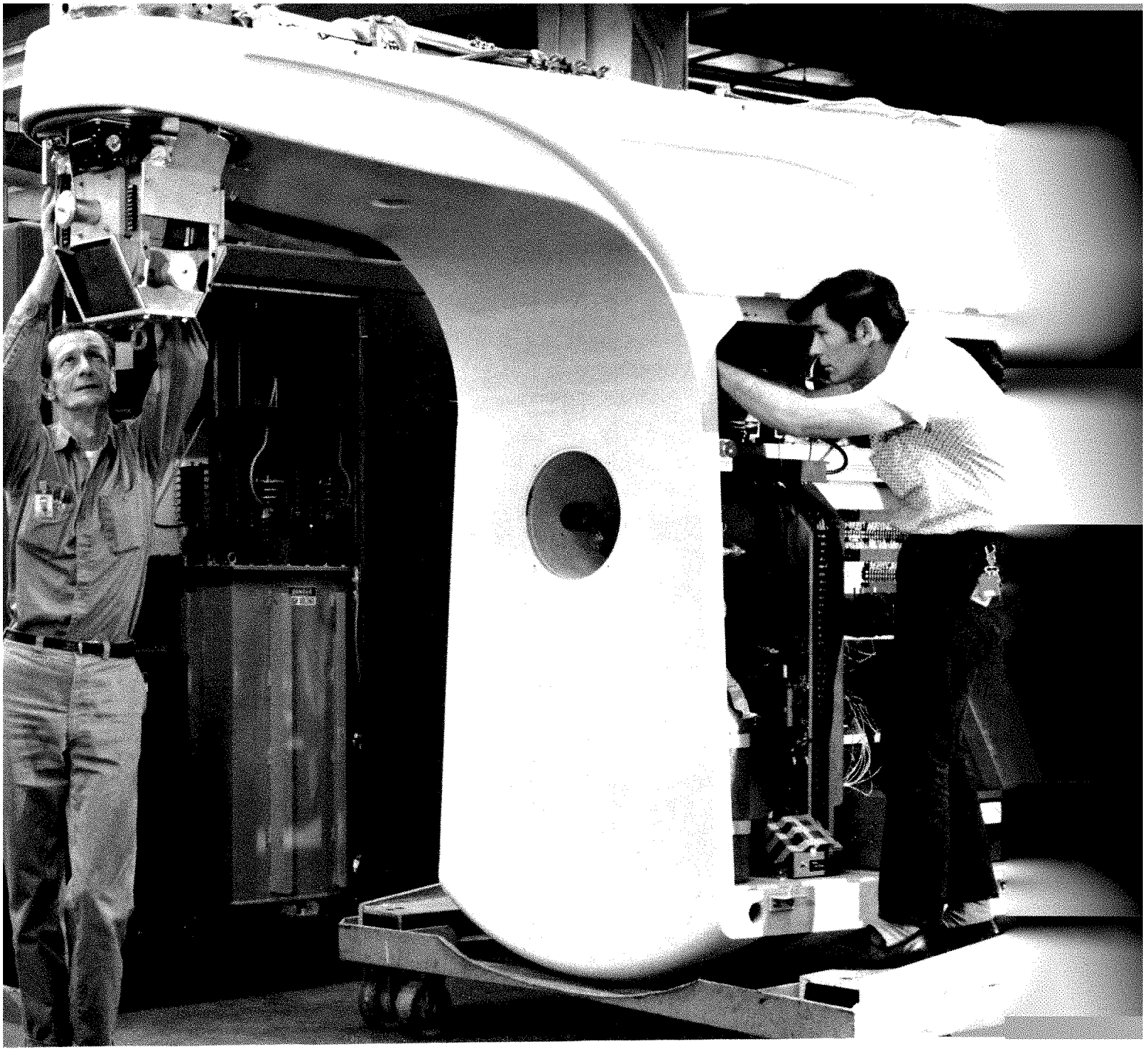
ANOTHER VARIAN CLINAC* 6 MEV

Varian's ninth Clinac is being constructed for the Hamilton Clinic of the Ontario Cancer Foundation. In addition to the eight Clinacs now in service, Varian has delivered an additional 14 linear accelerators in the United States and abroad. In the background is one section of waveguide, and the modulators for a 135 MEV linear accelerator being developed for the University of Saskatchewan, where it will be used for advanced photo-nuclear studies.

■ **The Clinac system produces greater than 400 rad/min/meter, flattened to ±3%. Focal spot is less than 3 mm. Leakage radiation outside of the main cone is less than 0.1% of central axis intensity. The X-ray head has beam-defining jaws which move on arcs subtending the target and provide a wide range of field sizes from zero to 27 x 30 cm at 1 meter.**

■ **The accelerator may be rotated 360° about the horizontal axis, while maintaining 1 meter between the target and the isocenter. No floor cutouts are required. Total variation of X-ray field flatness is maintained within ±3% during 360° machine rotation.**

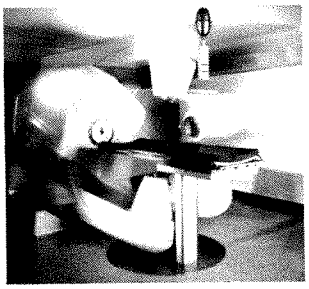
■ **The Clinac system has an overall height of seven feet, eight inches. It can be accommodated in a room with a ceiling height of eight feet. The complete system is self-contained, consisting of only the accelerator and the control console. No separate equipment rooms are required.**



RADIOTHERAPY LINEAR ACCELERATOR

■ **Specially-designed patient support assembly permits rapid positioning and treatment of large numbers of patients on a routine basis.**

Write for our comprehensive brochure on the Clinac system. Your inquiries are invited in relation to Varian's new deferred-payment plan for medical organizations. To demonstrate the Clinac accelerator in action, Varian will loan without charge a 16 mm sound/color motion picture



Gentlemen: Please ☐ Send me additional technical information. ☐ Send me the Clinac brochure. ☐ Send me the Clinac color/sound film. ☐ Have a Varian representative call on me. *Trademark

NAME & TITLE_____

HOSPITAL_____

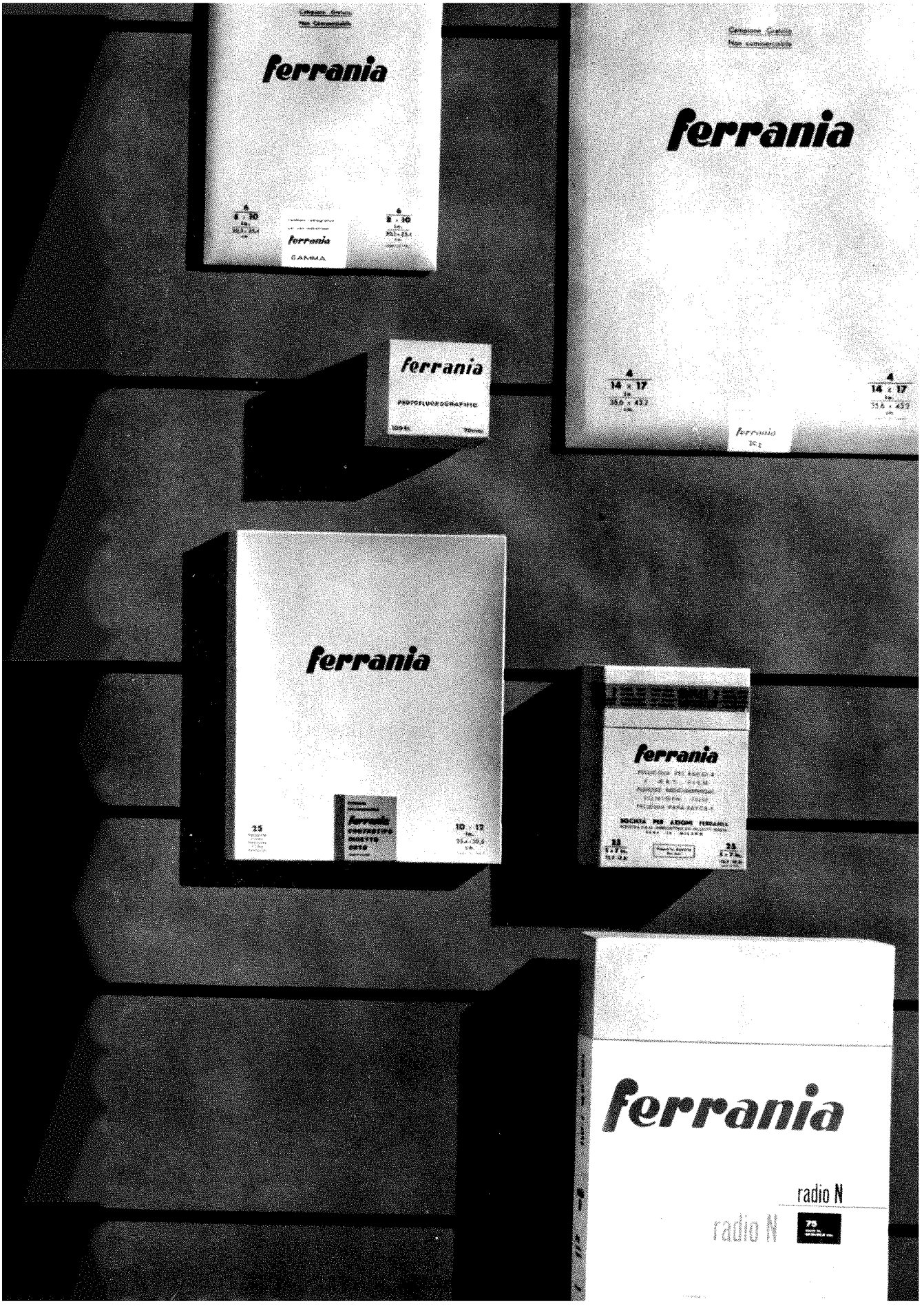
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TYPE N *for use with intensifying screens*

Speed: fast
Contrast: good
Grain: normal
Base: blue, safety

Type N film is faster than the most widely used medical X-ray film in the United States—and it costs less. This film is available with square corners in boxes of 25 or 75 interleaved sheets. For rounded corners, and extra savings on economical Ferrania prices, order Type N in bulk packages of 300-sheet interleaved, or 500-sheet non-interleaved films. Bulk-packed Type N film is specially designed for processing in all automatic developing units.

SIMPLEX *non-screen, for use with direct X-rays*

Speed: very fast
Contrast: good
Grain: fine
Base: blue, safety

No cassette is needed with Simplex film—each sheet is sealed in a lightproof exposure envelope for radiography of limbs and extremities. Convenient guide lines divide the upper surface of the envelope permitting accurate masking of the film into four separate exposure areas. 25 Simplex exposure envelopes are packaged per box.

GAMMA *for ultra fine tissue work*

Speed: slow
Contrast: extremely high
Grain: extremely fine
Base: blue, safety

This new Ferrania film brings unprecedented detail to mammography and other ultra-fine tissue radiography. Gamma film is compatible with all standard developing systems, and is packed 25 exposure envelopes per box.

IC_2 *for very fine tissue work*

Speed: medium
Contrast: high
Grain: very fine
Base: blue, safety

IC_2, slightly faster than Gamma film, permits additional savings for mammography and similar fine tissue work when the maximum requisites are high contrast and very fine grain. It is packed 25 exposure envelopes per box.

X2A and X3V *for photofluorography*

Speed: fast
Contrast: high
Grain: fine
Base: blue, safety

Ferrania films for photofluorography record all details of the smallest structures. X2A is highly sensitive to blue and violet light, and is used with blue fluorescing screens. X3V is orthochromatic, and is used with yellow-green fluorescing screens. Both films are available in 100- or 550-ft. rolls of 70mm-wide film, wound emulsion side in on cores.

DIRECT POSITIVE *for fast reproduction*

Speed: medium
Contrast: excellent
Grain: fine
Base: clear, safety

The special, clear safety base of this film permits an exceptional reproduction of any radiograph. None of the definition or contrast of the original will be lost. In fact, this film can actually be used to improve these factors. Direct positive film does not utilize intermediate copies and makes reproductions in as little as two minutes. It is packed in boxes of 25 interleaved sheets.

Seven *ferrania* X-ray films meet your needs for quality radiographs

Ferrania offers substantial savings on excellent medical X-ray films for all diagnostic applications. Name your requirement—screen or non-screen radiographs, fine tissue work, or photofluorography. Ferrania has six films of unmatched quality to fill these needs. The seventh, a high-contrast, direct positive film, reproduces even the finest details of a radiograph. Ferrania-formula chemicals, in addition, guarantee that the high contrast and clarity of Ferrania films will be retained during processing.

Every sheet and roll of film meets Ferrania's exacting standards of performance. This is true for the film you buy today, next month, or next year. Such consistency is the result of Ferrania's exacting quality control program—one that has supplied films of premium quality to the world for more than 35 years.

To find out more about Ferrania quality, test it. A request to Cenco X-Ray brings a test quantity of film (any of the seven types) and a catalog from your local Ferrania dealer. Also included: a copy of *Radiographica*, a technical journal of X-ray diagnosis published as a service by Ferrania.

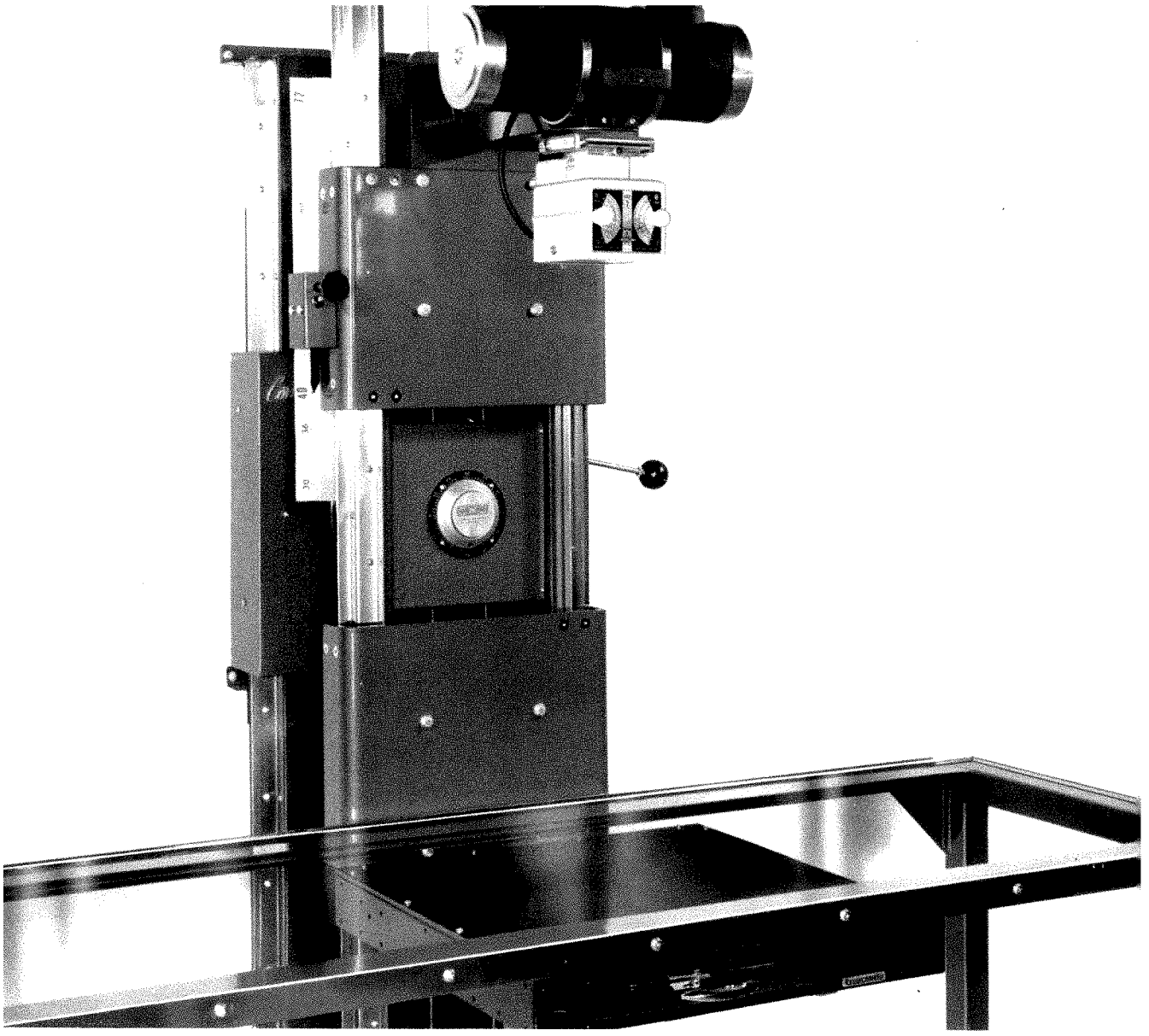
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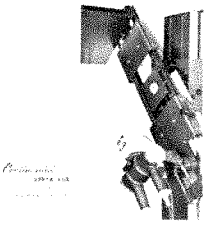
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for Versatile Radiographic Views

A radiographic unit *plus*—with remarkable speed and ease of use! A fully counter-balanced, bucky-tube positioning structure with INDEPENDENT ADJUSTMENT of:

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Conveniently handles a wide variety of conventional techniques *plus* many special techniques such as: Direct image magnification • Stretcher bucky radiography • Skull and sinuses.



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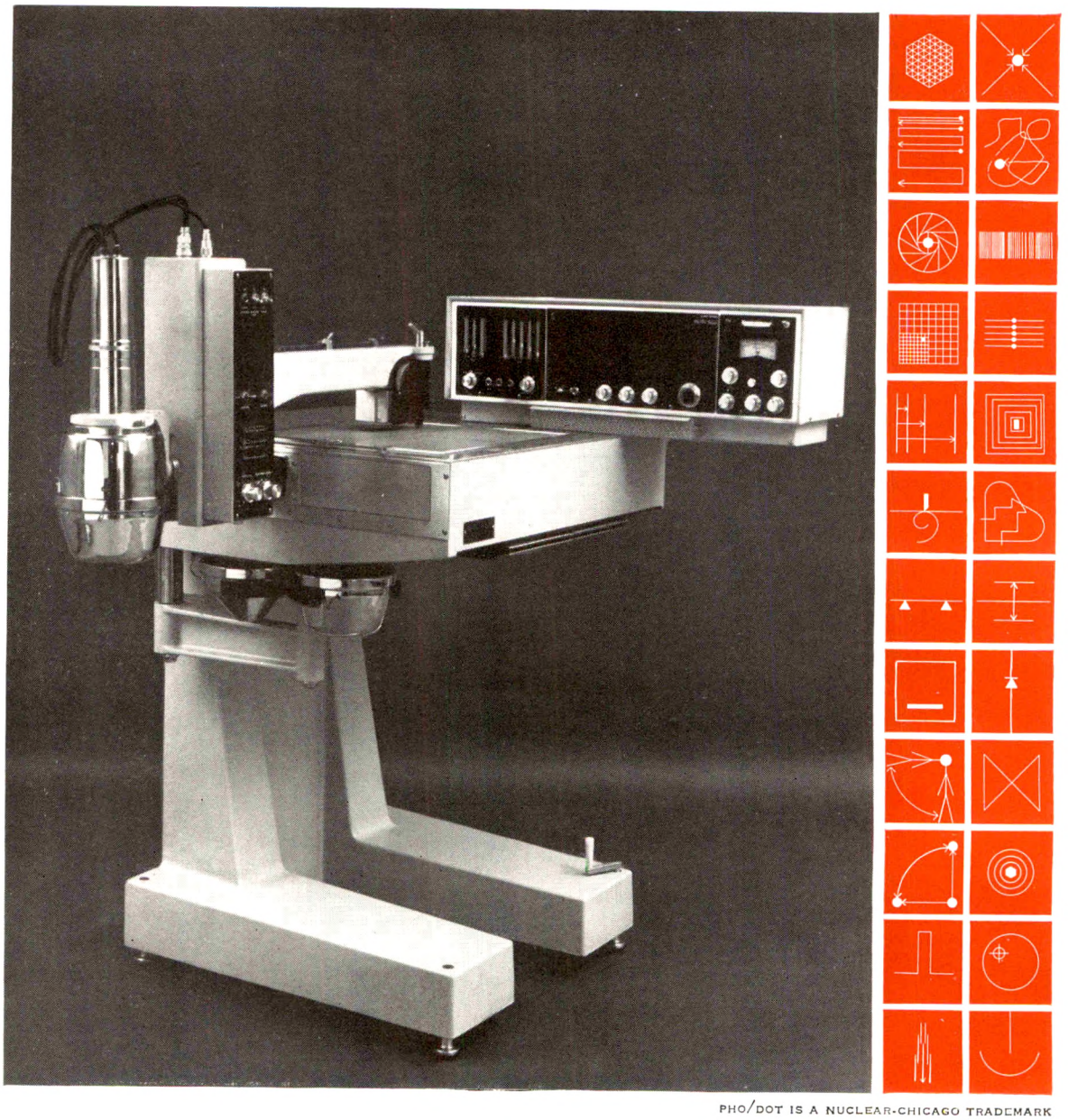
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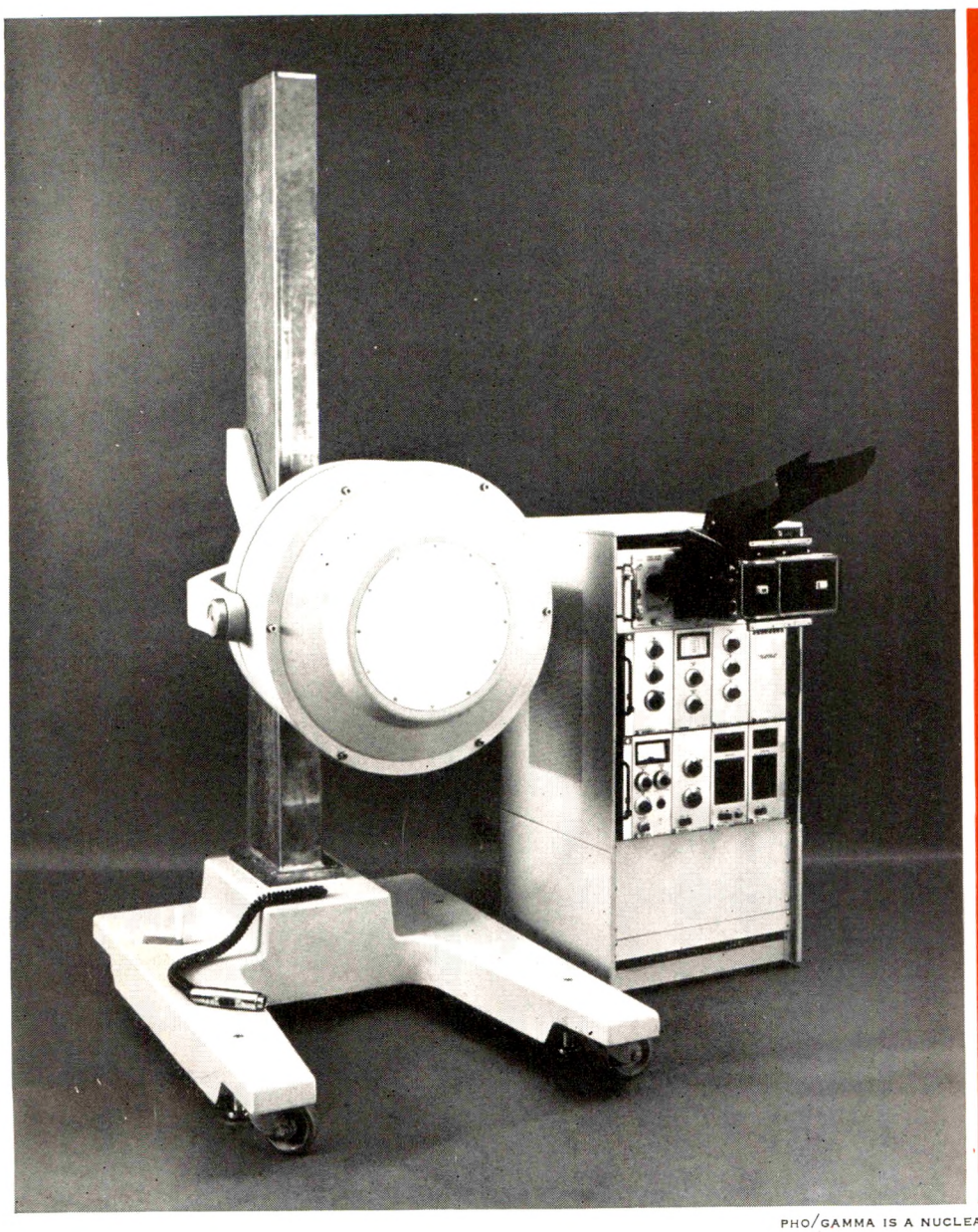
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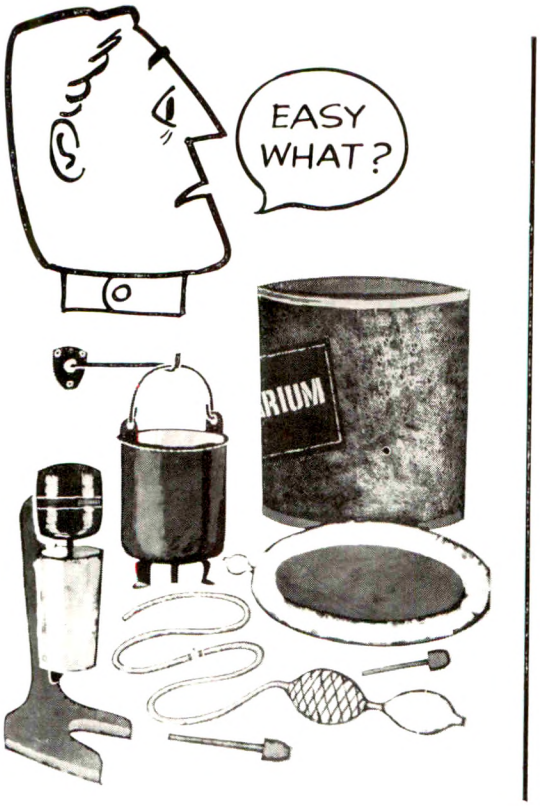
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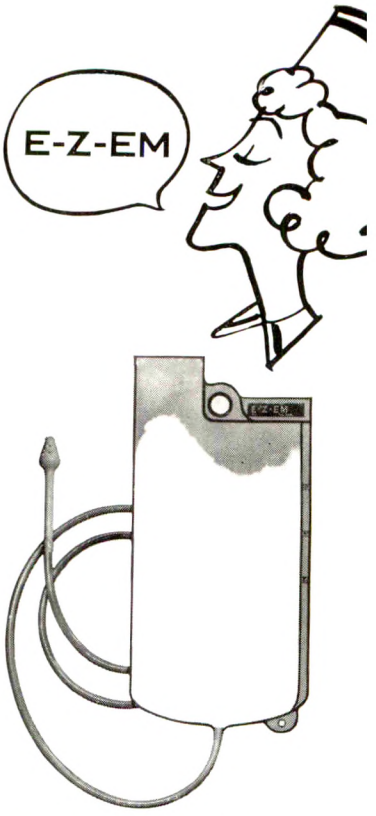
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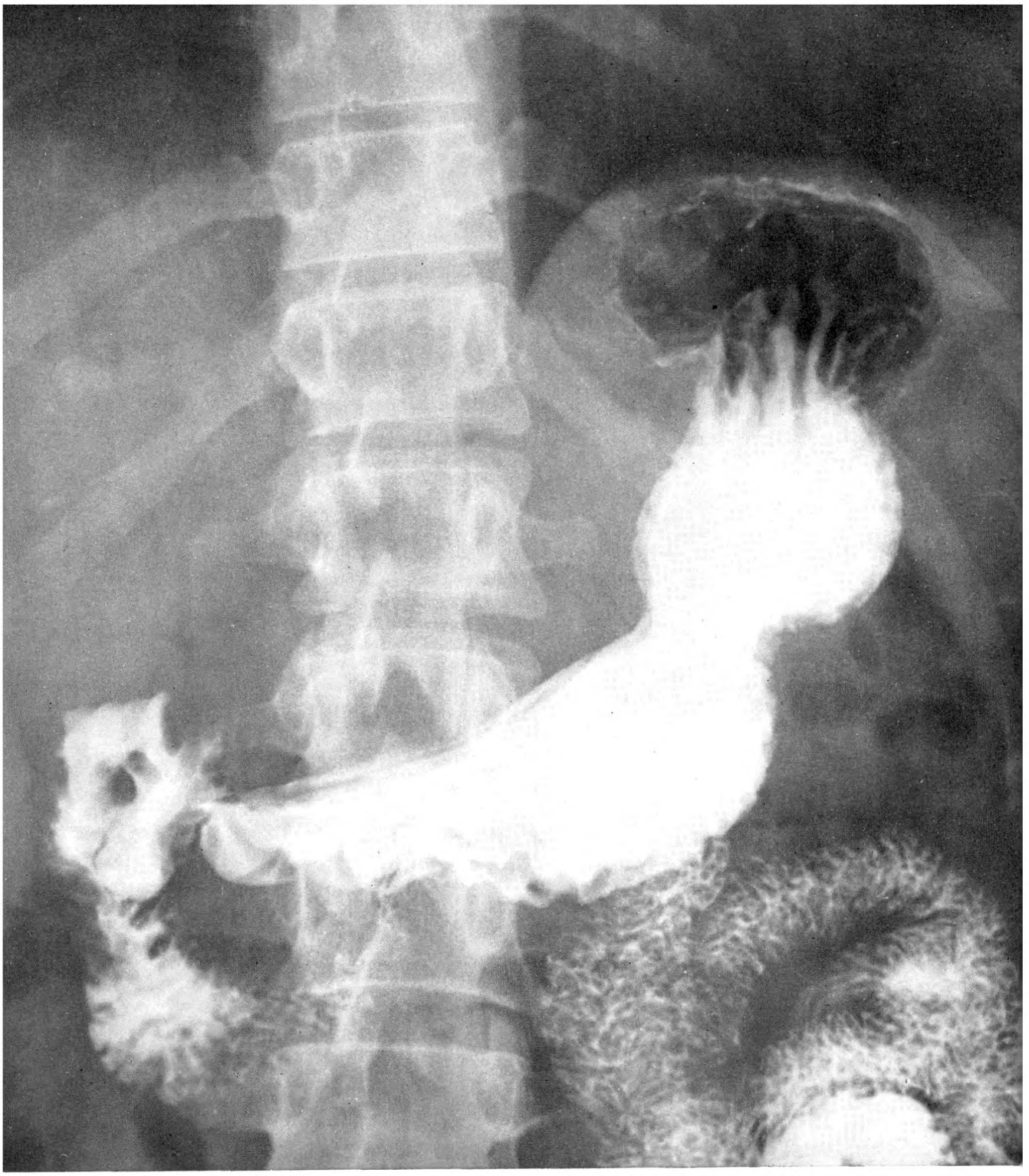
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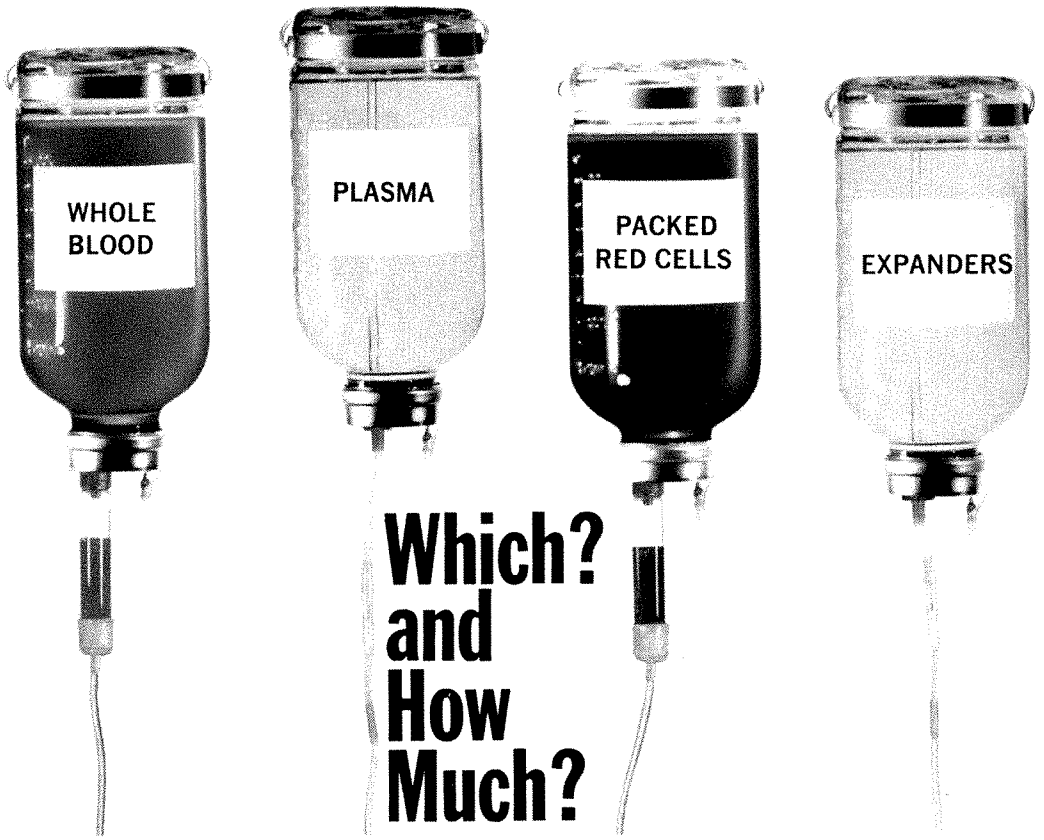
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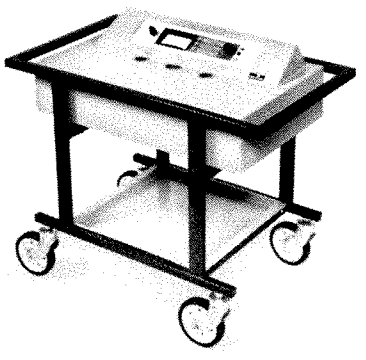


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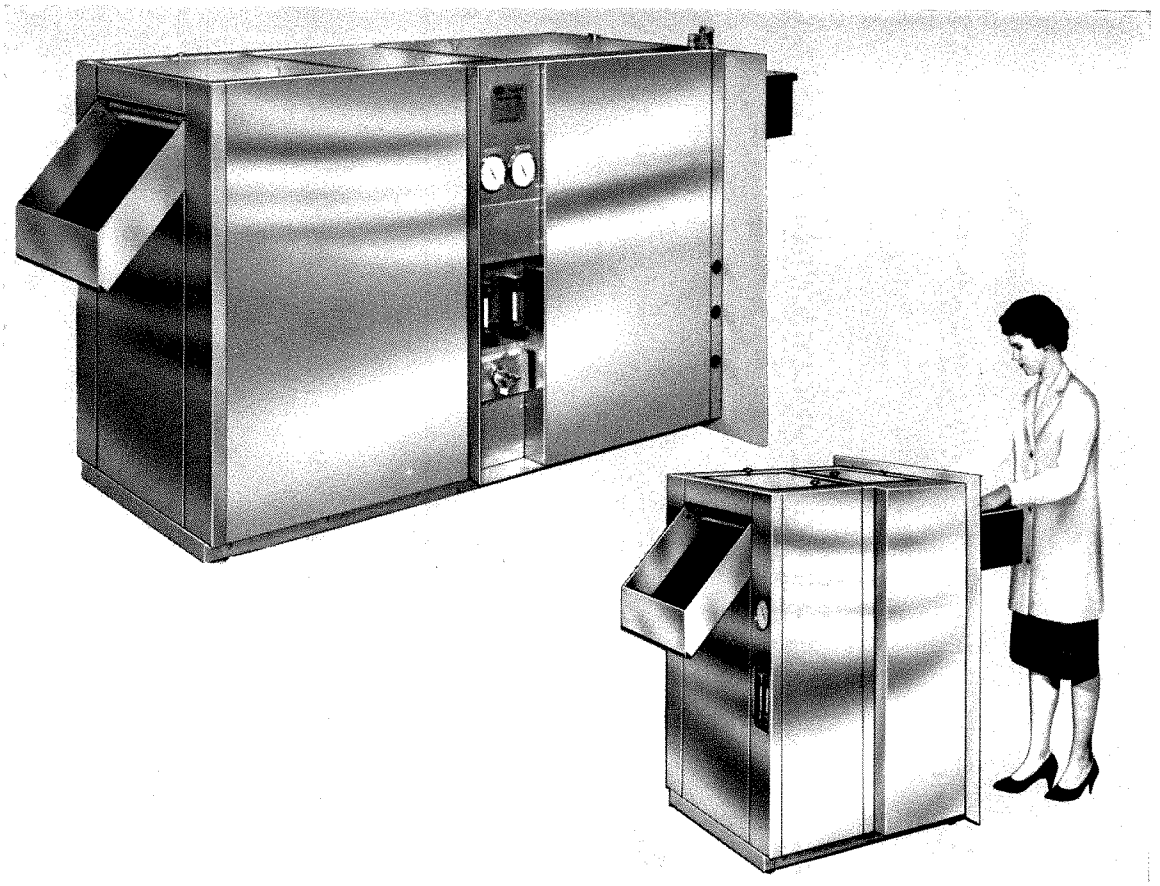
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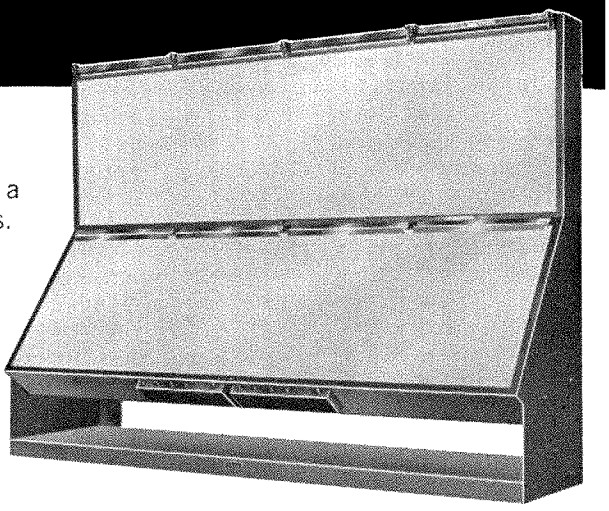
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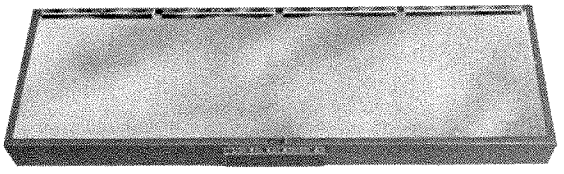
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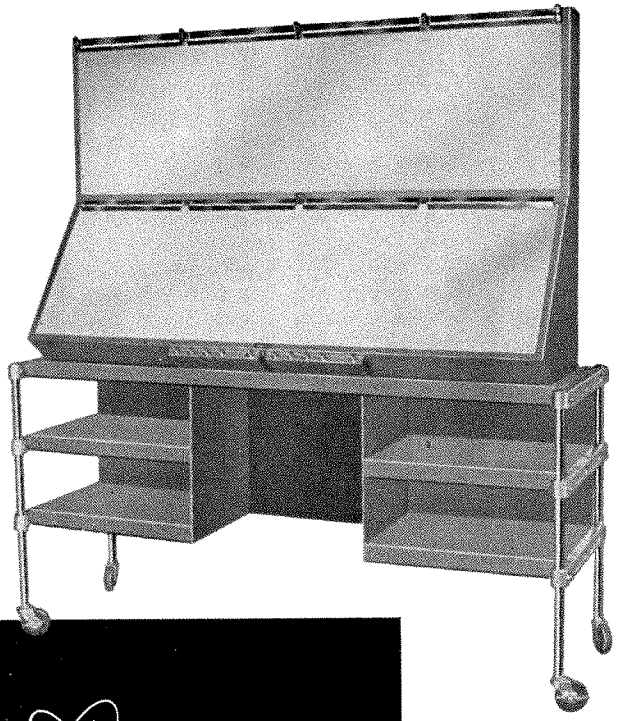
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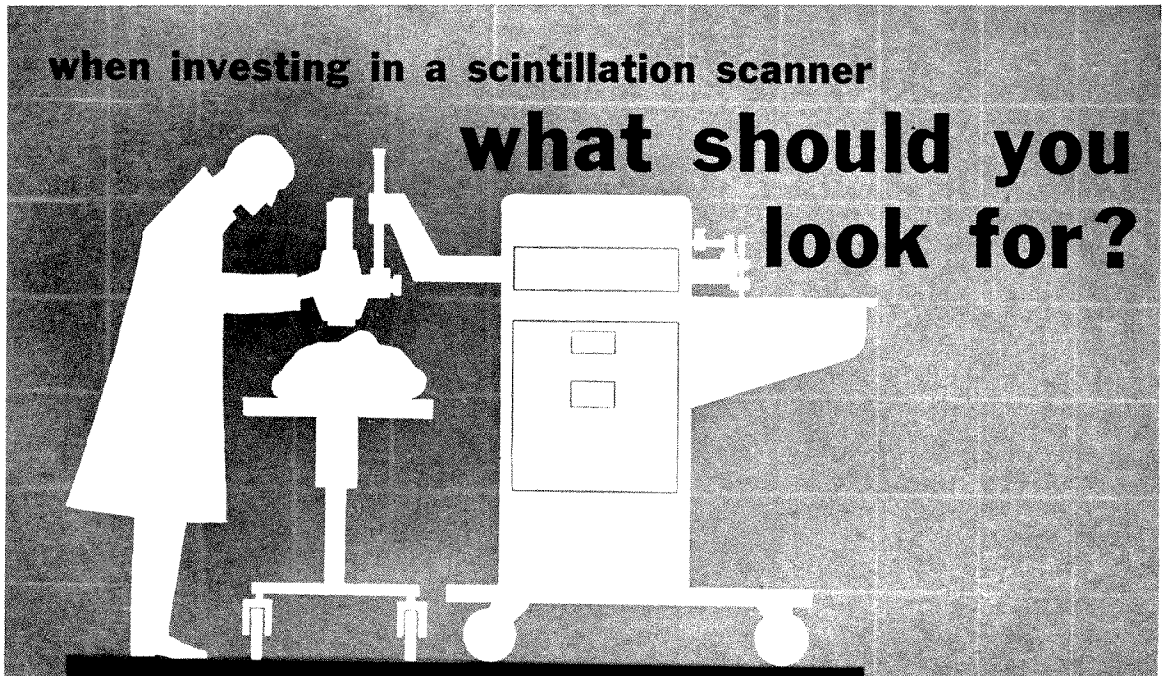
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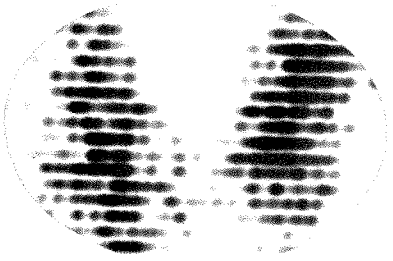
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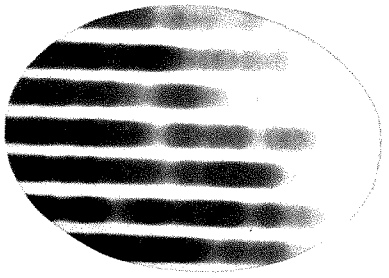


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VOL. 91 APRIL, 1964 No. 4

ROENTGEN ANATOMY AND TERMINOLOGY OF GASTRIC SURGERY*

By H. JOACHIM BURHENNE, M.D.
SAN FRANCISCO, CALIFORNIA

THE radiologic study is the most important procedure in the diagnosis of the postoperative stomach. In order to extract all the available information from this examination, the radiologist must fully understand an assortment of gastric operations (Table 1, Fig. 1 and 2). The large number of eponyms used, however, in the surgical literature and the lack of semantic uniformity are both complex and discouraging.

In 1939, Pólya[7] enumerated 49 typical methods available for the re-establishment of gastrointestinal continuity after stomach resection. He pointed out that the particular surgical procedure carrying his name was, in fact, performed earlier by 11 other well known surgeons, one of whom was Hofmeister. The "Hofmeister procedure," on the other hand, was practiced first by Eiselsberg. The custom of attaching a surgeon's name to a certain operative modification loses even more of its significance when we learn that the so-called "Hofmeister valve" was devised after his death, independently by both Lake and Finsterer.[6]

Nomenclature is useful when clear in definition and when universally acceptable. Terminology such as "partial gastrectomy" and "gastro-duodenostomy" is readily understood, whereas "Billroth 1" means little in countries where the same procedure is named after Péan and Rydigier. This particular operative procedure was, in fact, performed first by these latter two surgeons.

The existing dilemma was well exemplified by Ogilvie's[6] attempt to distribute surgical credit. He described the most satis-

TABLE I

BASIC COMPONENTS OF GASTRIC SURGERY

1. Extent of gastric resection
2. End or side anastomosis
3. Anterior or posterior anastomosis
4. Superior or inferior anastomosis
5. Large or small stomal diameter
6. Slow or rapid gastric emptying
7. Antecolic or retrocolic gastro-jejunostomy
8. Right-to-left or left-to-right direction of anastomosis
9. Short or long proximal jejunal limb
10. Horizontal or oblique plane of anastomosis
11. Direction of gastric emptying

* From the Department of Radiology, University of California School of Medicine, San Francisco, California.

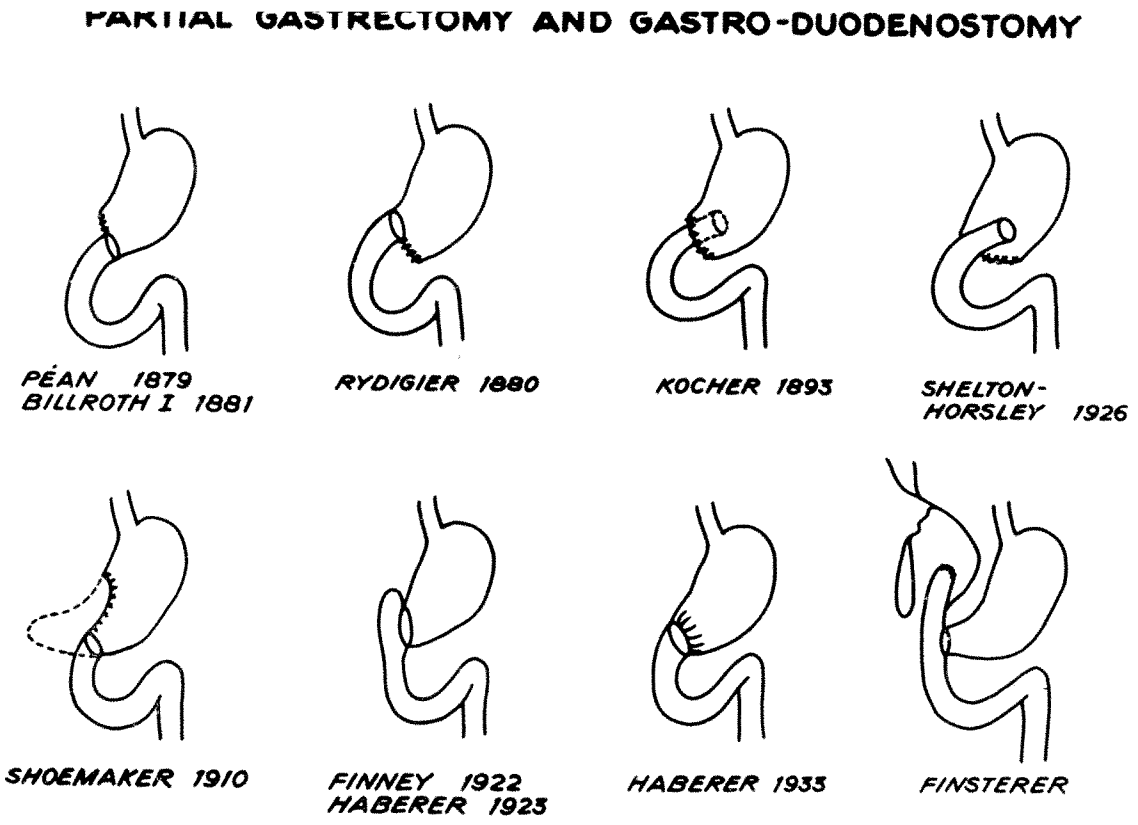


FIG. 1. Varieties of gastro-duodenostomy.

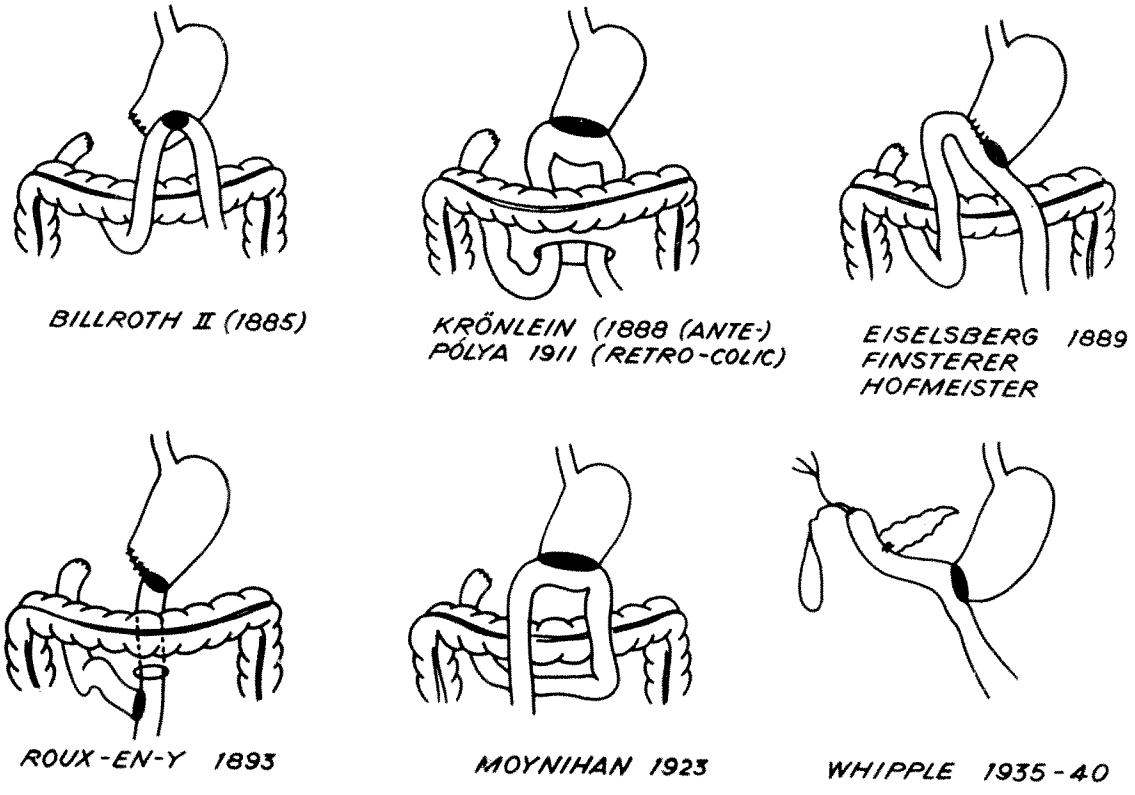


FIG. 2. Varieties of gastro-jejunostomy.

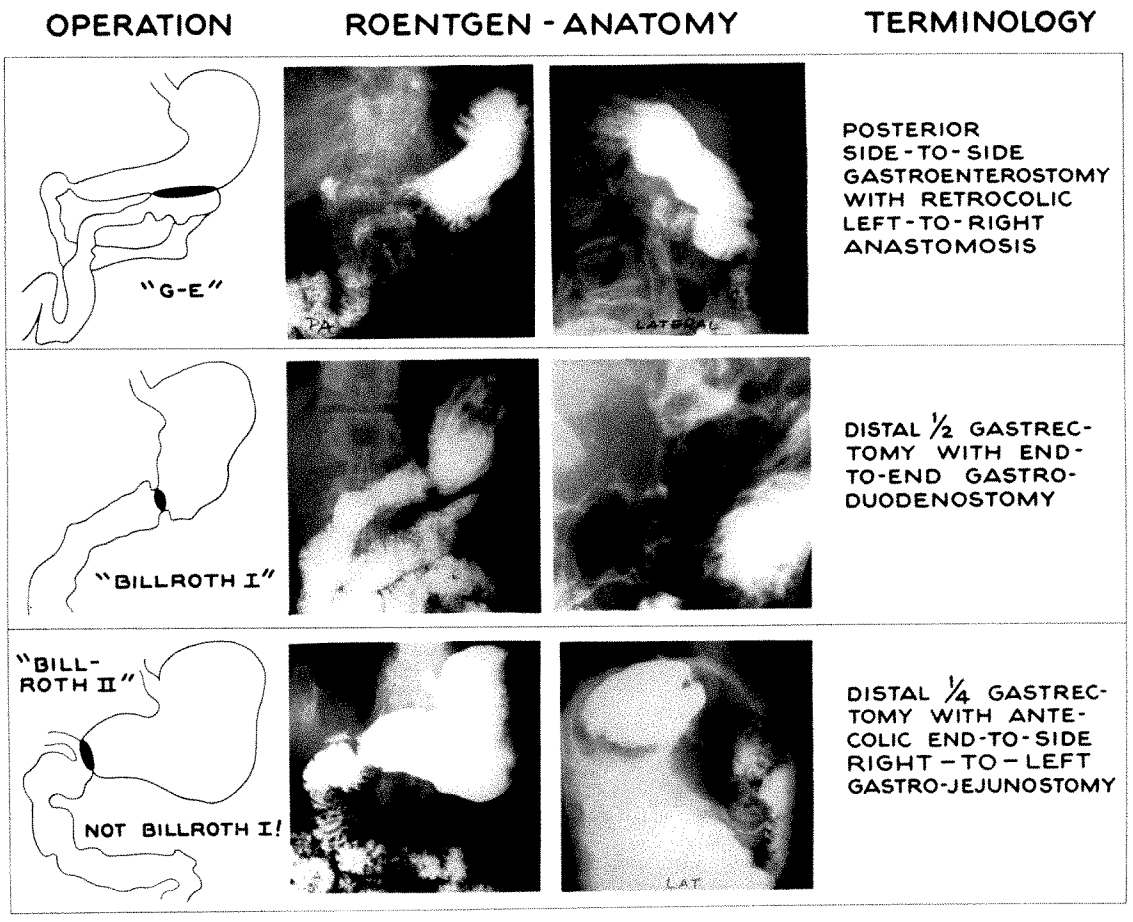


Fig. 3. When compared to eponyms, descriptive terminology is clear and understood widely.

factory gastrectomy as "the high posterior Finsterer-Lake-Lahey modification of the Miculicz-Kronlein-Hofmeister-Reichel-Polya improvement of the Billroth II gastrectomy with a large valve and a small stoma."

When eponyms are used, the information in a patient's record or in an operative or roentgenologic report is neither useful nor precise. We suggest that a purely descriptive terminology is best suited for all purposes. It defines clearly, may be applied to existing or future operative modifications, and is understood widely (Fig. 3 and 4).

COMPONENTS OF GASTRIC SURGERY

If all variants of gastric surgery are analyzed, 11 basic components become clear (Table 1). Any combination of these variants will determine a different surgical procedure. The knowledge of these building stones is fundamental for the understanding of postoperative anatomy and mechanics.[8] Furthermore, an analysis of these components by roentgenologic techniques represents the best diagnostic evaluation of the postoperative status.

1. EXTENT OF GASTRIC RESECTION (Fig. 5)

If the surgeon aims at removal of the acid-bearing portion of the stomach, usually a resection of the distal two-thirds or three-quarters is performed with the inclusion of a sleeve of the duodenal bulb. If the surgical objective is to resect the gastrin-producing portion of the stomach, an antrectomy or distal hemigastrectomy, including a duodenal sleeve, is performed.

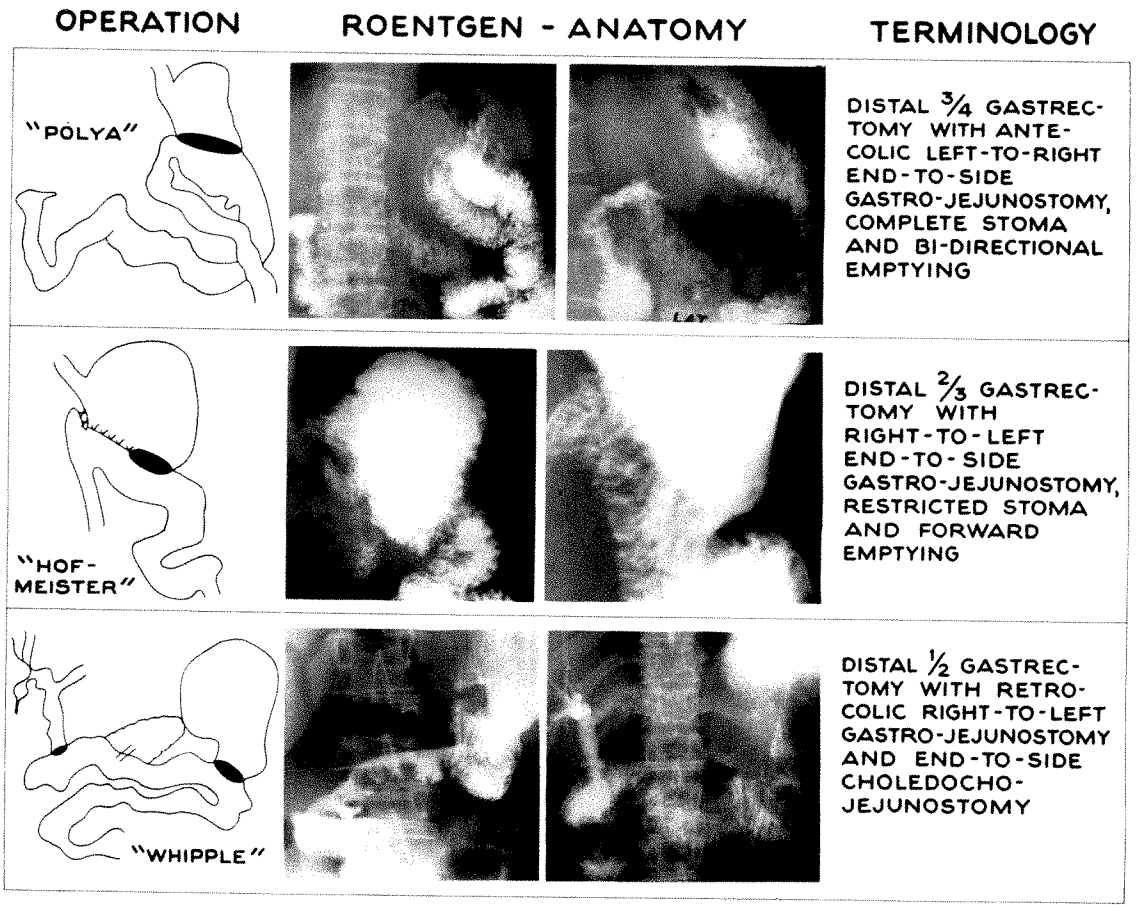


Fig. 4. Descriptive terminology is best suited for all purposes.

Woodward[9] has pleaded for more accurate use of the term "subtotal" gastrectomy, employed much too freely in "partial" gastrectomies. An estimate based on the roentgenograms of the amount of stomach remaining after resection may be misleading because the remnant enlarges with delayed gastric emptying and its size decreases with mechanical dumping.

2. END OR SIDE ANASTOMOSIS (Fig. 6)

The four different types of anastomosis possible are described as end-to-end anastomosis, end-to-side anastomosis, side-to-side anastomosis, or side-to-end anastomosis. The original partial gastrectomy and gastro-

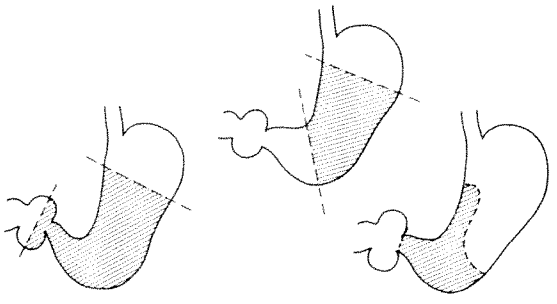


Fig. 5. Extent of gastric resection.

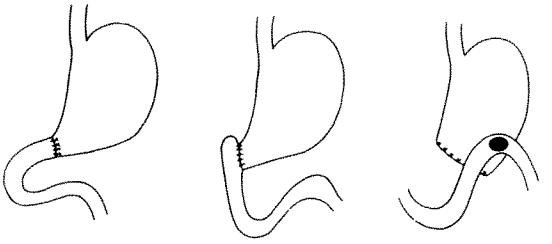


Fig. 6. End or side anastomosis.

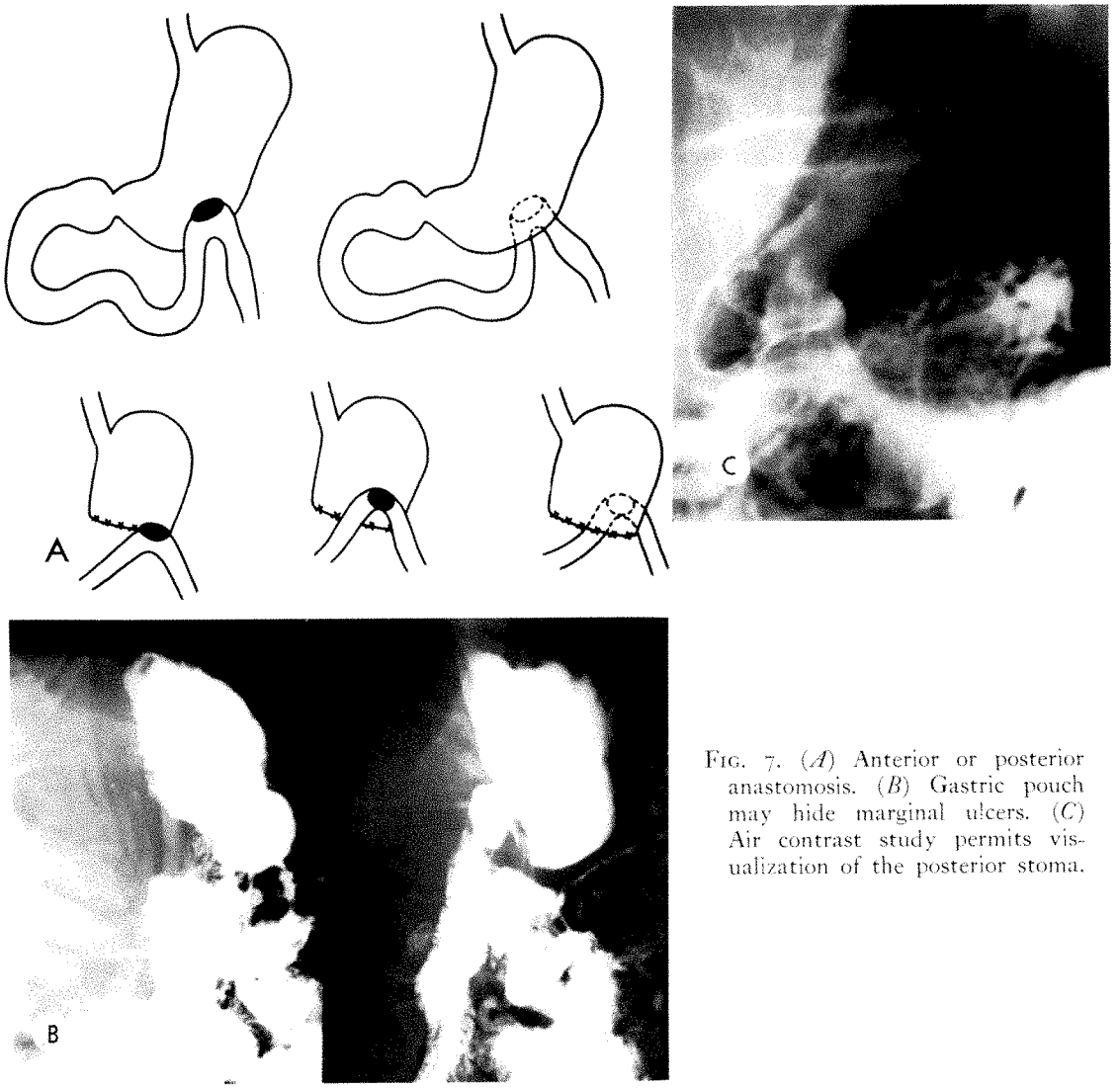


Fig. 7. (*A*) Anterior or posterior anastomosis. (*B*) Gastric pouch may hide marginal ulcers. (*C*) Air contrast study permits visualization of the posterior stoma.

jejunostomy was performed as a side-to-side anastomosis (Billroth II), whereas today an end-to-side anastomosis is customary (Fig. 2).

3. ANTERIOR OR POSTERIOR ANASTOMOSIS (FIG. 7 *A*)

It is important for the radiologist to realize that the stoma is not always incorporated into the stomach at its cut end, but that it may be placed into the anterior or posterior wall of the stomach. Therefore, the gastric pouch may hide marginal ulcers (Fig. 7 *B*). If the opening lies on the posterior wall, superimposition of air in the stomach will permit visualization of a good air contrast of the stoma itself (Fig. 7 *C*).

4. SUPERIOR OR INFERIOR ANASTOMOSIS (FIG. 8 *A*)

The restricted stoma may be incorporated into the cut end of the stomach superiorly to the right at the lesser curvature (Fig. 8 *B*) or inferiorly to the left at the greater curvature. Depending on its placement, the preference of gastric emptying into either the proximal or distal limb will be influenced.

5. STOMAL SIZE (FIG. 9)

The entire cut end of the stomach may



Fig. 8. (*A*) Superior or inferior anastomosis. (*B*) Superior anastomosis is rarely performed.

be utilized for a *complete* stoma, the so-called "Pólya" gastrectomy. For a *restricted* stoma (so-called "Hofmeister" gastrectomy), the cut end of the stomach is partially closed.

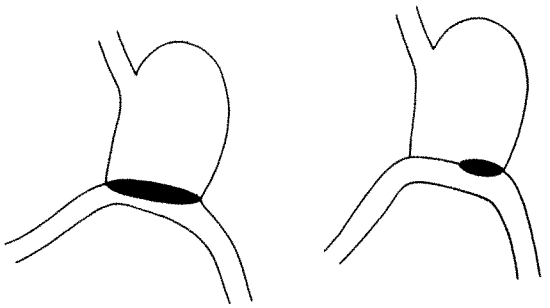


Fig. 9. Stomal size.

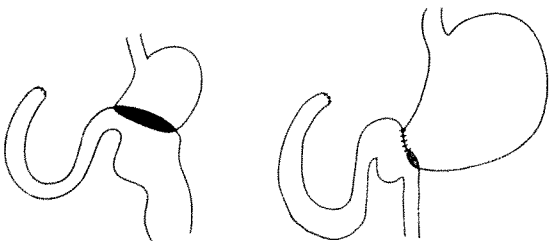


Fig. 10. Gastric emptying time and reservoir function.

6. Gastric emptying time (Fig. 10)

The normal gastric reservoir is lost in all patients after gastrectomy, and rapid emptying into the jejunum is present to some degree. The gastric emptying time is particularly accelerated with the more extensive gastric resection and in the presence of a large stoma.[1,5] Surgical revision of a large stoma into a small one, or conversion from a gastro-jejunostomy into a gastro-duodenostomy has restored better function of the reservoir.[4] Kirklin[3] pointed out that the rugae in the gastric remnant may appear accentuated because of rapid emptying and incomplete distention of the remaining stomach pouch.

7. Antecolic or retrocolic gastro-jejunostomy (Fig. 11)

The jejunum may be placed in front of the colon or behind it through a surgical opening in the mesocolon. Roentgenologically, its placement is best distinguished in the oblique and lateral projections. If the colon is outlined by gas, better estimation of position is possible. In the antecolic anastomosis, the proximal jejunal limb usually is longer than in the retrocolic.

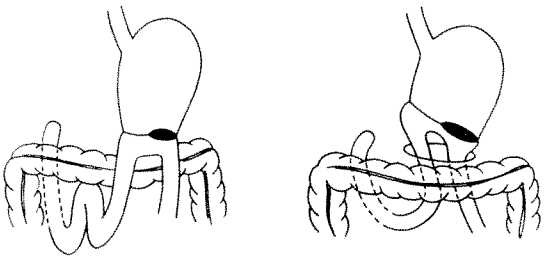


Fig. 11. Antecolic or retrocolic gastro-jejunostomy.

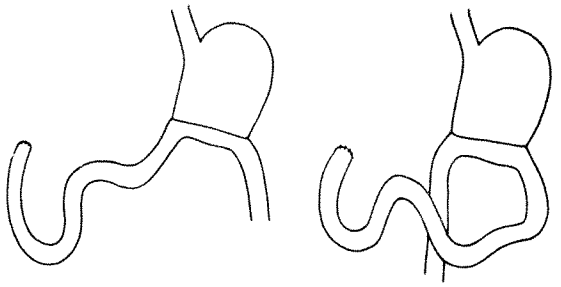


Fig. 12. Right-to-left or left-to-right direction of anastomosis.

8. RIGHT-TO-LEFT OR LEFT-TO-RIGHT ANASTOMOSIS (FIG. 12)

The anastomotic loop consists of a *proximal* (afferent) and *distal* (efferent) jejunal limb. The proximal limb coming from the duodenum will lead to the lesser curvature in a right-to-left gastro-jejunal anastomosis. In this case, the distal limb leaves from the greater curvature (aniso-peristaltic or antiperistaltic). Conversely, the proximal jejunal limb is attached to the greater curvature of the stomach in a left-to-right anastomosis. The distal jejunal limb leads away from the lesser curvature (isoperistaltic).

If the proximal jejunal limb fills, the position of the ligament of Treitz and the duodenal loop are the best landmarks for its identification. Usually, the distal jejunal limb follows the remainder of the small bowel in a forward manner. Also, the direction of peristalsis, which is best studied with cineroentgenography, helps to identify the type of anastomosis performed.

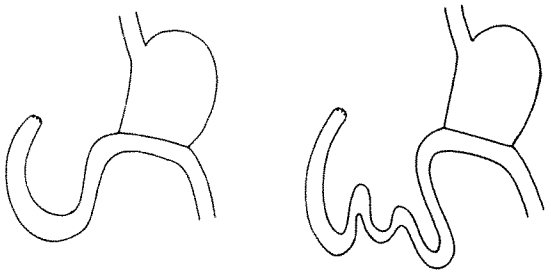


Fig. 13. Length of proximal jejunum.

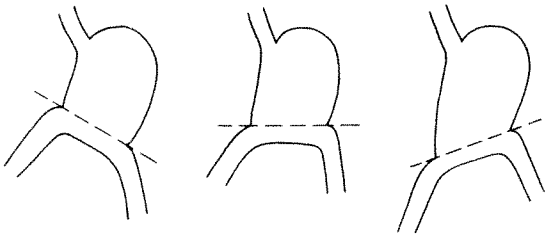


Fig. 14. Plane of anastomosis.

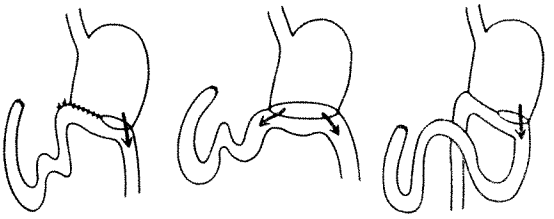


Fig. 15. Direction of emptying.

9. SHORT OR LONG PROXIMAL JEJUNAL LIMB (FIG. 13)

An antecolic anastomosis usually requires a longer proximal jejunal limb. If it fills and is demonstrated roentgenologically, the length of the proximal limb should be stated in the report. Long proximal limbs are more frequently associated with post-gastrectomy complications.



Fig. 16. Gastrostomy with feeding tube.

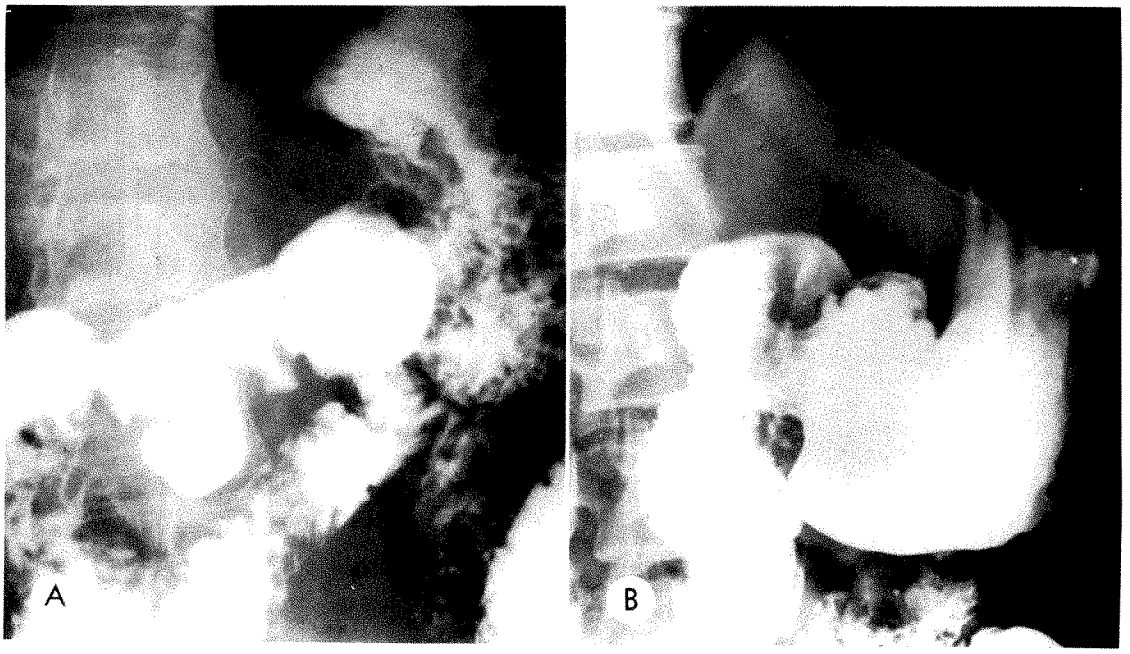


Fig. 17. (*A*) Pseudodiverticulum deformity usually seen with pyloroplasty (Heineke-Mikulicz). (*B*) Gastroduodenostomy (pyloroplasty) (Finney). Note the more vertical arrangement of the duodenum following its mobilization (Kocher).

10. HORIZONTAL OR OBLIQUE ANASTOMOSIS (FIG. 14)

The anastomotic plane is important in the determination of the direction of gastric emptying. Most surgeons perform an oblique anastomosis with more extensive gastric resection along the lesser curvature.



Fig. 18. Distortion of the fundus after wedge resection for benign gastric tumor.

Combined with an inferiorly located stoma, this anastomosis will result in preferential emptying into that jejunal limb which has been attached to the greater curvature.

11. DIRECTION OF EMPTYING (FIG. 15)

The factors listed under 4, 5, 8, and 10 influence the jejunal direction of gastric emptying. The operation with the best functional result is a one-way gastrectomy with emptying into the distal jejunal limb.[2] Filling of the proximal jejunal limb and even preferential gastric emptying into it do not necessarily imply a malfunctioning and symptomatic gastro-jejunostomy. It is distention and stasis with delayed emptying of the proximal limb and duodenum that indicate a pathophysiologic situation.

DISCUSSION

In medical practice, such words as "efferent" and "afferent" are as impractical as eponyms. They require frequent spelling for clarification, whereas "proximal" and "distal" are easily understood. One is tempted to forget the ambiguous terms

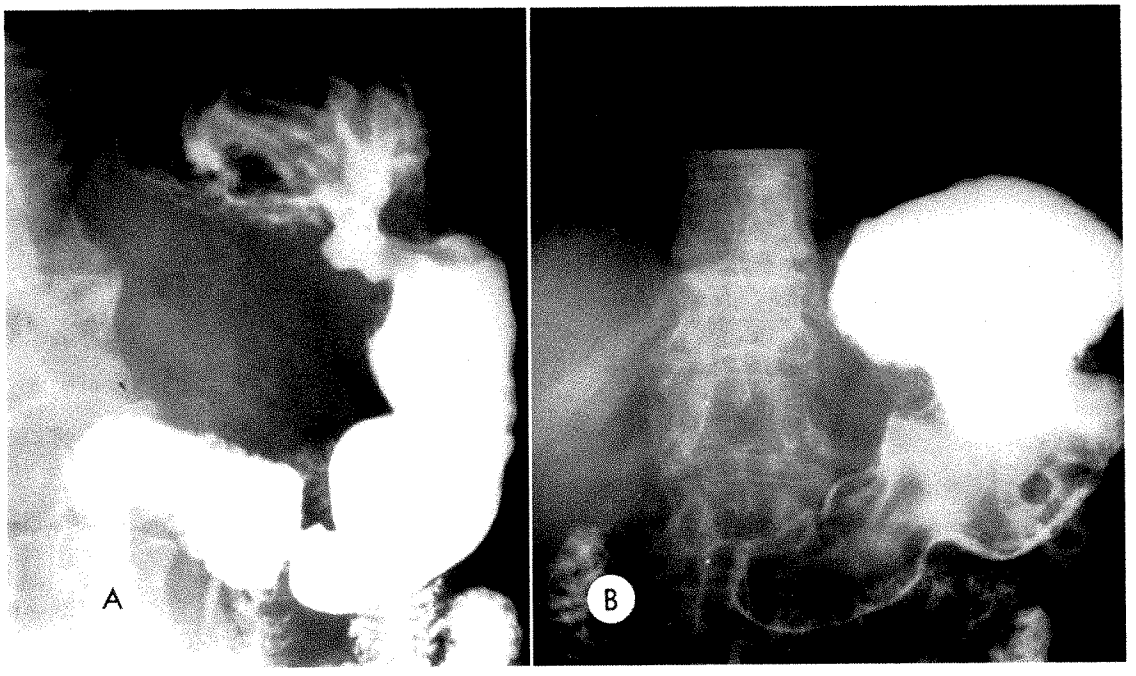


Fig. 19. (*A* and *B*) Segmental resection for gastric ulcer with end-to-end gastro-gastrostomy and large ulcer recurrence.

"isoperistaltic" and "antiperistaltic" in favor of the clear definition "left-to-right" or "right-to-left" anastomosis. The radiologist, however, must be familiar with the common language of gastric surgery. He has to master both eponyms for consultation and clear descriptive terminology for his reports. Only close consultation between the surgeon and the radiologist allows the extraction of the best possible diagnosis from the roentgenologic study.

Because of geographic variation in usage, no universally acceptable list of eponyms can be collected. The more common

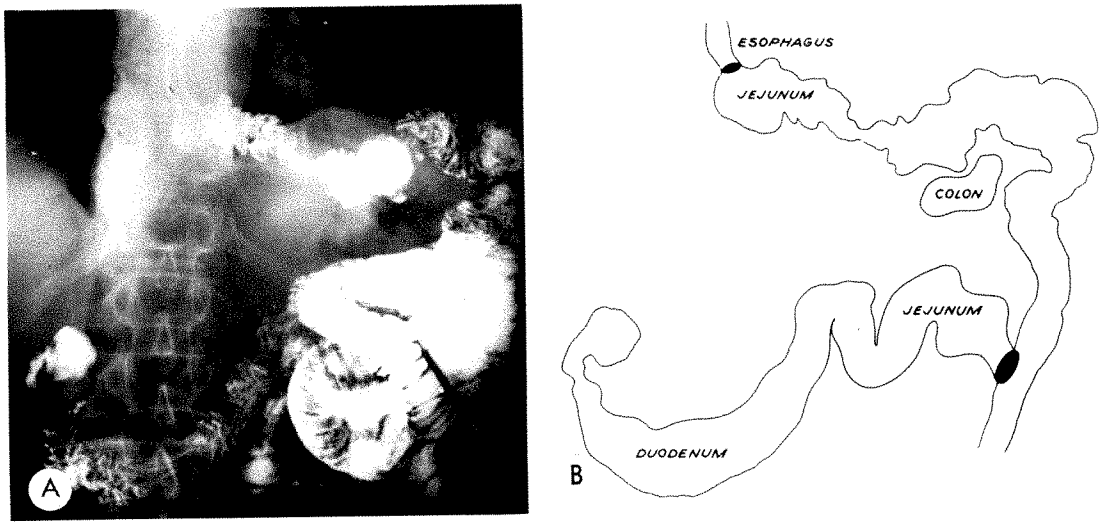


Fig. 20. (*A* and *B*) Total gastrectomy and antecolic, end-to-end esophago-jejunostomy with end-to-side jejuno-jejunostomy ("Roux-en-Y").

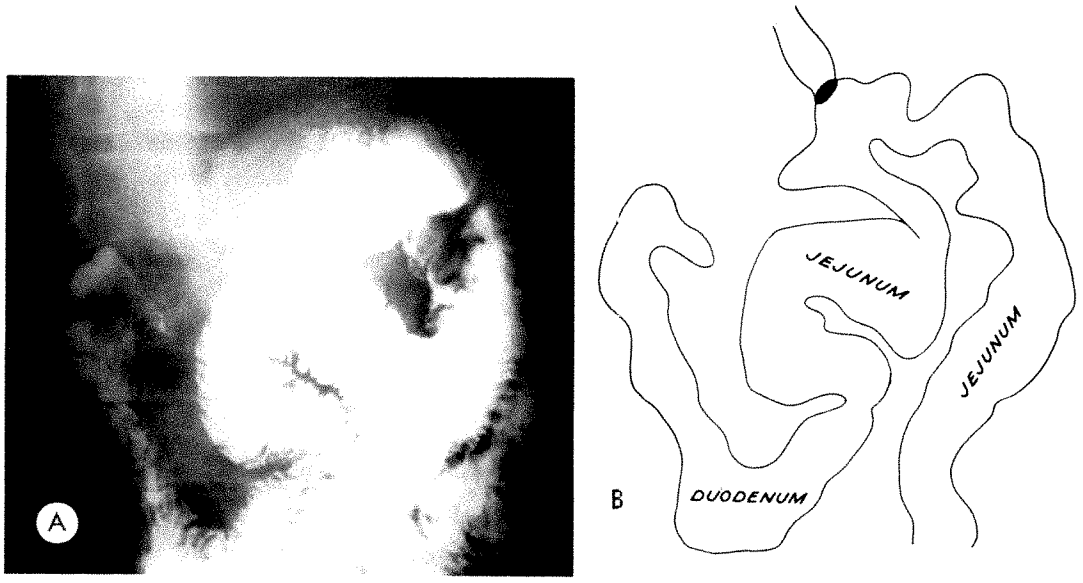


FIG. 21. (*A* and *B*) Total gastrectomy and end-to-side esophago-jejunostomy.

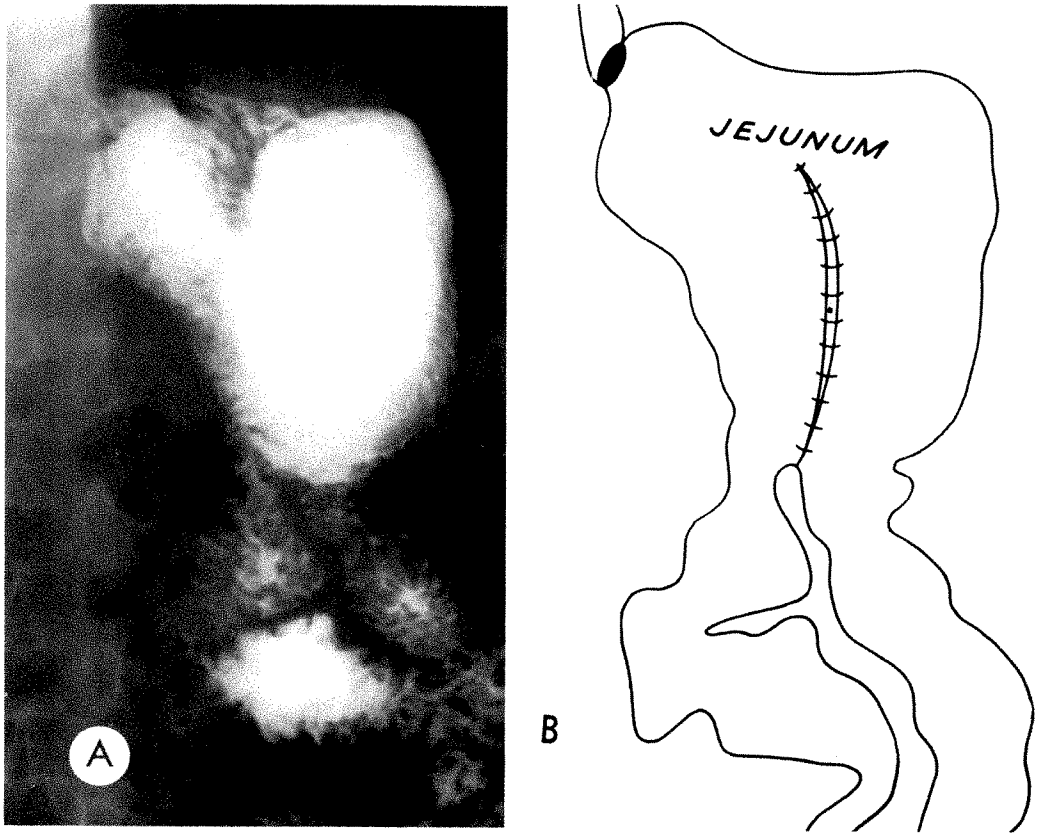


FIG. 22. (*A* and *B*) Total gastrectomy and end-to-side esophago-jejunostomy with side-to-side jejuno-jejunostomy for attempted restoration of reservoir.

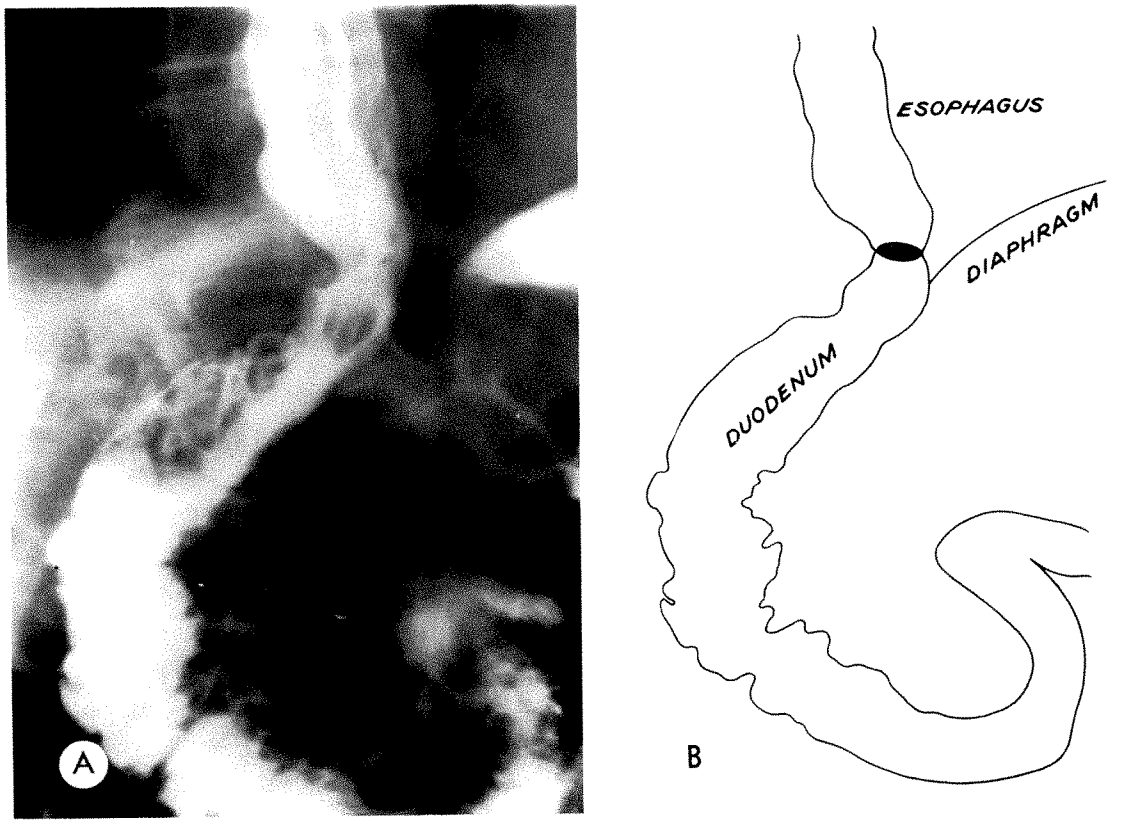


Fig. 23. (*A* and *B*) Total gastrectomy and end-to-end esophago-duodenostomy.

eponyms used in North America would be defined as follows:

Billroth I—Any partial gastrectomy with gastro-duodenostomy.

Billroth II—All varieties of partial gastrectomy and gastro-jejunostomy.

Pólya—Any gastro-jejunostomy using the entire cut end of the stomach for the anastomosis.

Hofmeister—A gastro-jejunostomy with a restricted stoma and partial closure of the cut end of the stomach. At present, the names of Pólya and Hofmeister are used independently whether the anastomosis is antecolic, retrocolic, left-to-right, or right-to-left.

Roux-en-Y—Any end-to-side jejuno-jejunostomy with or without anastomosis with the esophagus, stomach, duodenum, gallbladder, bile duct, or pancreas.

Most roentgenologic examinations of the gastrointestinal tract are performed with the patient's history lacking. Therefore, routine questioning about previous operations is necessary before contrast studies are begun. Even when the clinician is aware of previous gastric surgery in a patient, information concerning the particular operative procedure may not be obtainable. If the various described components of gastric surgery are familiar to the radiologist, reconstruction of the performed operation is possible in most cases.

(*Please turn page for Figures 24 and 25*)

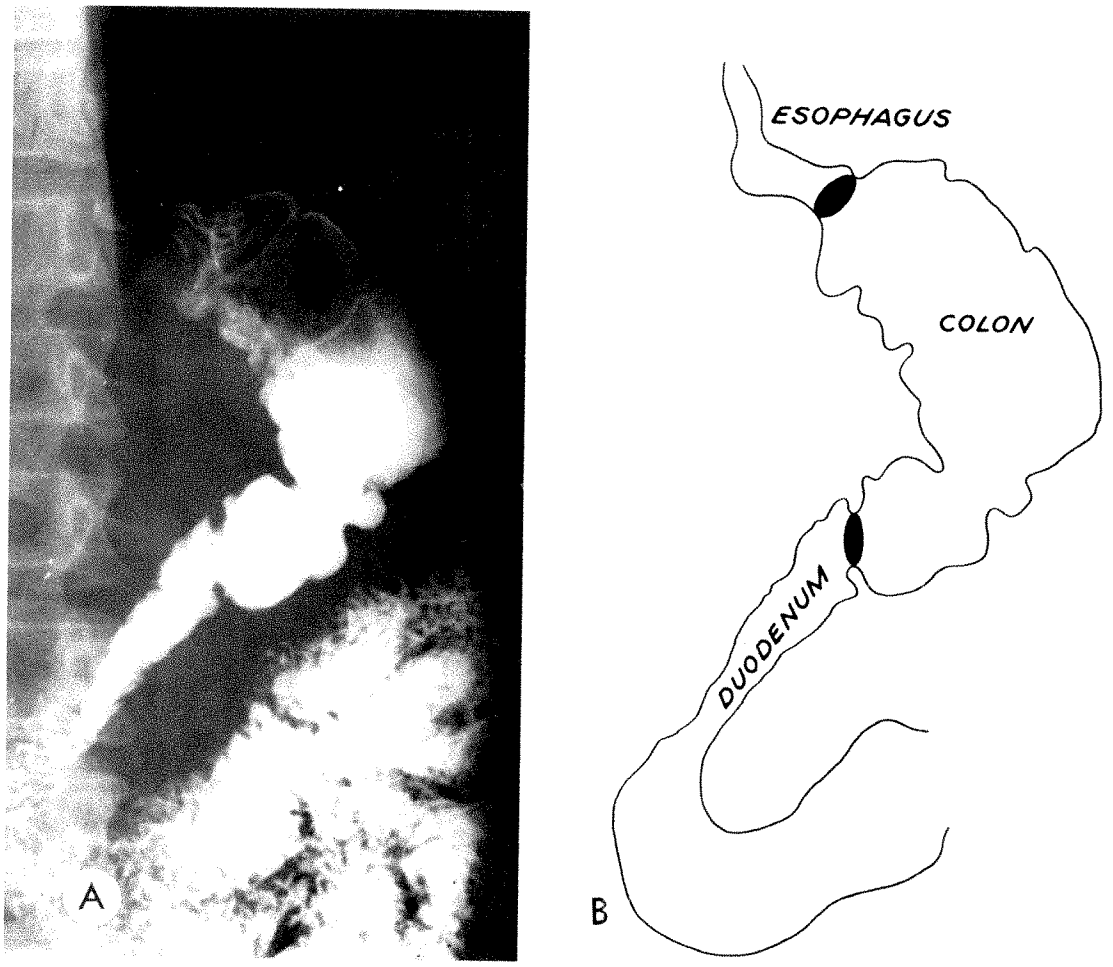


Fig. 24. (*A* and *B*) Total gastrectomy and colonic interposition.

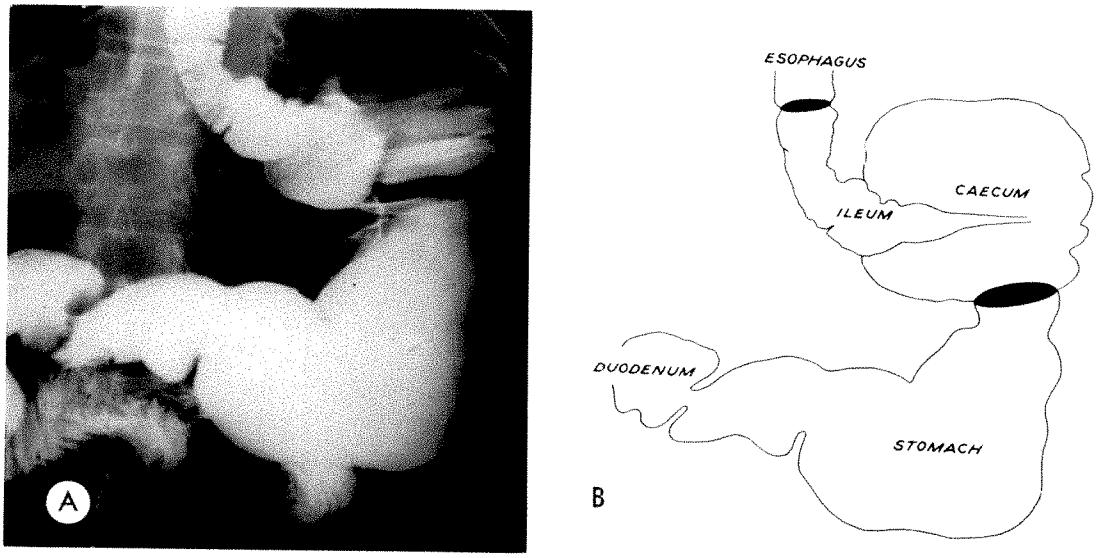


Fig. 25. (*A* and *B*) Ileocecal interposition in partial gastrectomy.

TABLE II

A SYSTEMATIC ARRANGEMENT OF GASTRIC OPERATIONS ACCORDING TO THEIR MORPHOLOGIC ALTERATIONS

1. *No gastric resection—no anastomosis*
   - Gastrotomy—for exploration
   - Gastrostomy—for feeding or decompression (Fig. 16)
   - Gastropexy or gastrorrhaphy—for ptosis, volvulus, and herniation
   - Simple excision—for ulcer
   - Simple closure—for perforation
   - Gastroplasty—for stenosis
   - Pylorotomy—for stenosis (Ramstedt-Frèdet, 1912)
   - Pyloroplasty—for drainage (Heineke, 1886; Mikulicz, 1888) (Fig. 17 *A*)
   - Vagotomy—for ulcer therapy, complete or selective

2. *Enteroanastomosis—no gastric resection*
   - Gastro-duodenostomy—for pyloric obstruction and drainage (Finney, 1902) (Fig. 17 *B*)
   - Gastro-enterostomy—for ulcer or obstruction; may be anterior, posterior, antecolic, retrocolic, right-to-left anastomosis, or left-to-right anastomosis (Fig. 3, top) (Wölfler, 1881)

3. *Partial gastric resection—no enteroanastomosis*
   - Wedge resection—for ulcer, biopsy, or benign lesion (Fig. 18)
   - Segmental resection—for ulcer or tumor (Fig. 19, *A* and *B*), (Mikulicz, 1897)

4. *Partial gastric resection and enteroanastomosis*
   - Gastrectomy with gastro-duodenostomy (Fig. 1)—may be end-to-end, end-to-side, side-to-end, or side-to-side with superior, inferior, posterior, or anterior anastomosis
   - Gastrectomy with gastro-jejunostomy (Fig. 2)—may be end-to-side, side-to-side, or end-to-end, with full or restricted stoma, retrocolic or antecolic, right-to-left or left-to-right anastomosis

5. *Total gastrectomy*
   - Esophago-jejunostomy—may be end-to-end (Fig. 20, *A* and *B*), or end-to-side (Fig. 21, *A* and *B*; and 22, *A* and *B*) (Schlatter, 1897)
   - Esophago-duodenostomy—may be end-to-end (Fig. 23, *A* and *B*) (Brigham, 1898)
   - Interposition operation—utilizing small or large bowel (Fig. 24, *A* and *B*) or ileocolic segment (Fig. 25, *A* and *B*). Also may be performed with partial gastrectomy

Note: Illustrative examples typifying some of the above mentioned procedures are shown in Figures 16 through 25 inclusive.

SUMMARY

The radiologist must be familiar with all types of gastric operations (Table II).

An analysis of surgical procedures of the stomach according to the described 11 basic components is the most helpful for the roentgenologic diagnosis of postoperative anatomy and postgastrectomy complications.

In roentgenology of the postoperative alimentary tract, descriptive terminology serves the best purpose. Eponyms transmit little useful information.

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GASTRIC ULCER IN THE AGED AND CALCIFIED ATHEROSCLEROSIS*

By ARTHUR ELKELES, M.D., F.F.R., R.C.S.I., D.M.R.E.
LONDON, ENGLAND

IT IS widely accepted that hyperacidity of the gastric juice plays an important part in the development of duodenal ulcer, but its role in the pathogenesis of gastric ulcer is still uncertain. While it is assumed that the hyperacidity associated with duodenal ulcer is of nervous origin, it has been suggested that an augmented hormonal phase of gastric secretion is responsible for the development of the lesser curve gastric ulcer. Dragstedt,[3,4] supported by much clinical and physiologic evidence, has shown that stasis in the antrum of the stomach leads to increased gastrin production with subsequent parietal cell stimulation. He arrived at the conclusion that delayed emptying of the stomach, either of organic or functional origin, is responsible for the development of the lesser curve gastric ulcer. This concept offers a plausible explanation for those gastric ulcers which occur in connection with pyloric channel disease. However, in the classic solitary lesser curve ulcer, neither pyloric channel disease nor any appreciable delay in emptying of the stomach are usually found. Important features of these ulcers are normal or hyposecretion of hydrochloric acid, a small parietal cell mass, and a large alkali secreting zone. These ulcers tend to develop in advancing years. They are often large, and giant ulcers are not uncommon.

When neither pyloric channel disease nor hyperacidity of the gastric juice seem to play a part in the development of these ulcers, could they be due to an increased vulnerability of the gastric mucosa caused by an impaired blood supply? The most frequent cause of circulatory disturbances in advancing years is arteriosclerosis.

In previous investigations, Elkeles[6,7,10] found that patients with chronic gastric ulcer, aged over 50 years, show a significantly higher incidence of calcified atherosclerosis of the abdominal aorta than patients with duodenal ulcer and control cases of the same age groups. In conformity with these findings is a recent report by Holstein and Stecken.[14,15] They found that of 21 patients between the ages of 60 and 80 years in whom calcification of the abdominal aorta and of the left gastric artery could be seen on roentgenograms, 18 showed gastric ulceration.

In contrast to the high incidence of calcified atherosclerosis in patients with gastric ulcer, a very low incidence was found in patients with carcinoma of the stomach. This observation has been confirmed by Doll,[2] Jones and Gummer,[16] Dungal and Benediktsson,[5] and Fotopoulos *et al.*[12]

Roentgenography of the abdominal aorta can be used as a routine method for the detection of calcified atheromatous lesions. It is generally recognized that the earliest and the most advanced manifestations of general arteriosclerosis are found in the abdominal aorta. Moreover, the abdominal aorta and the celiac axis are the central arteries for the blood supply of the stomach. Although the roentgenographic method is limited to the detection of calcified atheroma, most of the advanced lesions contain sufficient calcium to be recognized on roentgenograms.

In this paper further evidence is presented for the concept that a close relationship exists between gastric ulcer in the aged and calcified atherosclerosis of the abdominal aorta and the celiac axis.

MATERIAL

This was a study of 2,185 patients, aged over 50 years, in whom the abdominal aorta

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TABLE I

CALCIFICATION OF THE ABDOMINAL AORTA IN PATIENTS WITH CHRONIC GASTRIC ULCER AND IN CONTROL CASES

Age Group (yr.)	Gastric Ulcer Group		Control Group		Difference (gastric ulcer-control)
	No. of Cases	Per Cent with Calcification	No. of Cases	Per Cent with Calcification	
Males					
50–60	100	65.0	295	23.4	41.6± 5.5
61–70	68	79.4	370	41.9	37.5± 6.6
71+	23	91.3	204	55.9	35.4±10.8
Females					
50–60	25	40.0	252	13.9	26.1± 7.7
61–70	42	78.6	304	48.0	30.6± 8.2
71+	36	100.0	279	73.1	26.9± 7.5

was roentgenographed. It comprises 293 patients with chronic benign gastric ulcer, of whom 191 are males and 102 females; 1,704 control cases, of whom 869 are males and 835 females; and 188 patients with cancer of the stomach. In the gastric series the abdominal aorta was roentgenographed prior to the barium meal examination regardless of the clinical diagnosis. The control series consists of unselected patients from in- and out-patient departments of general hospitals. The majority came from the orthopedic, physiotherapy or casualty departments.

A classification of the calcified atheromatous lesions has been attempted by designating a linear plaque approximately 3–5 cm. in length as +, 10–12 cm. or parallel linear shadows as ++, and more extensive lesions involving almost the entire abdominal aorta as +++.

RESULTS

THE INCIDENCE AND SEVERITY OF CALCIFIED ATHEROSCLEROSIS (TABLE I THROUGH IV)

The incidence and the severity of calcified lesions in the abdominal aorta are significantly higher in patients with gastric ulcer than in controls. This difference is highly significant in the 50 to 60 year age group, where 65 per cent of patients with gastric ulcer show calcified atheroma against 23.4 per cent in control patients. A striking difference in the incidence and severity of calcified atherosclerosis of the abdominal aorta is found between patients with gastric ulcer and those with carcinoma of the stomach. The application of this observation for the roentgenologic differential diagnosis between benign and malignant lesions of the stomach and its wider significance for the cancer problem have been referred to in previous publications.[8,9,11]

AGE AND SEX DISTRIBUTION

The present investigation reveals that the age distribution of the male patients with gastric ulcer is significantly different from the female age distribution: $X^2 = 30.332n' = 2p < 0.000001$. The peak incidence of gastric ulcer in males occurs between the ages of 50 to 60 years and that in females approximately 10 years later. Although the incidence of gastric ulcer is greater in males than in females up to the age of 70, the rate of increase in the incidence of gastric ulcer is much higher in females than in males after the age of 60.

It is of interest to point out that the age and sex distribution of gastric ulcer, found in this investigation, is similar to that of coronary heart disease. Oliver[19] reported that the peak incidence of ischemic heart

Table II

PERCENTAGE WITH SEVERE CALCIFICATION (++ AND +++) OF THE ABDOMINAL AORTA IN GASTRIC ULCER AND CONTROLS

Age Group (yr.)	Males			Females		
	Gastric Ulcer	Controls	Difference	Gastric Ulcer	Controls	Difference
50–60	48.0	13.2	34.8± 4.8	28.0	7.5	20.5±6.1
61–70	60.3	21.9	38.4± 5.9	59.5	36.5	23.0±8.0
71+	73.9	38.2	35.7±10.9	94.4	62.7	31.7±8.4

disease in women between 60 to 65 years is 5 or 10 years after the peak incidence in men. Although there are more male deaths from coronary heart disease at all ages, the rate of increase in coronary heart disease in women is 3 times that in men between 40 to 69 years. Peel,[21] who investigated the age and sex factors in 694 males and 171 females with coronary heart disease, found the peak incidence in males in the age group of 55 to 59 years; thereafter, the incidence falls almost as steeply as it rose. In females, however, the rise is steady almost in a straight line from age 40 to 70 years.

A similar age and sex distribution has been reported in the incidence of atherosclerosis and its cerebral complications in the South African Bantu. Laurie and Woods[18] found that after the age of 55 years, women are more prone to cerebral vascular disease with and without hypertension than males.

Assuming that coronary artery disease, cerebral vascular disease and gastric ulcer in the aged are complications of atherosclerosis, we would expect that their age and sex distribution corresponds to that of atherosclerosis. An analysis of the control series in Tables I and II shows that up to the age of 60 years, the incidence and the severity of calcified atherosclerosis of the abdominal aorta are higher in males than in females. Thereafter, this sex ratio is reversed, and the incidence and the severity of calcified atherosclerosis rise much more steeply in women than in men. Thus, the age and sex distribution of coronary heart disease, of cerebral vascular disease and of gastric ulcer tends to follow the age and sex distribution of calcified atherosclerosis.

CALCIFIED PLAQUES IN THE UPPER ABDOMINAL AORTA AND IN THE CELIAC AXIS

When the distribution of the calcified lesions in the abdominal aorta was studied more closely, it was found that in the majority of patients with gastric ulcer, independent of the extent and the degree of the atheromatous lesions, calcified plaques

Table III

CALCIFICATION OF THE ABDOMINAL AORTA IN GASTRIC ULCER AND IN CARCINOMA OF THE STOMACH (MALES AND FEMALES)

Age Group (yr.)	Gastric Ulcer		Gastric Carcinoma		Difference
	No. of Cases	Per Cent with Calcification	No. of Cases	Per Cent with Calcification	
50–60	125	60.0	52	1.9	58.1±8.2
61–70	110	79.1	80	10.0	69.1±7.3
71+	59	96.6	56	21.4	75.2±9.1

could be recognized in the upper abdominal aorta and in the region of the celiac axis. Figures 1 through 6 demonstrate the calcified lesions found in this series of patients with gastric ulcer.

DISCUSSION

The results of this investigation indicate that patients with gastric ulcer do not only show evidence of general arteriosclerosis but also of calcified atheroma in the central arteries supplying the stomach. Long ago Hauser[13] and other pathologists were impressed by the frequent association of gastric ulcer with general arteriosclerosis. They based the concept of the arteriosclerotic pathogenesis of these ulcers largely on this finding. From extensive clinical investigations, Spang[22] also arrived at the conclusion that gastric ulcer in the aged is the result of circulatory disturbances caused by general arteriosclerosis.

In spite of these contributions, the arteriosclerotic pathogenesis of gastric ulcer has not been widely accepted. The main argument against this concept has been the observation that, when partial gastrectomy was performed for peptic ulcer, the gastric and duodenal blood vessels of the resected specimens often failed to show evidence of local arteriosclerosis. Moreover, in the



Fig. 1. Fairly large lesser curve gastric ulcer. A second small ulcer is demonstrated by manual pressure. Note the densely calcified linear plaque in the abdominal aorta opposite the ulcers. Calcification+.

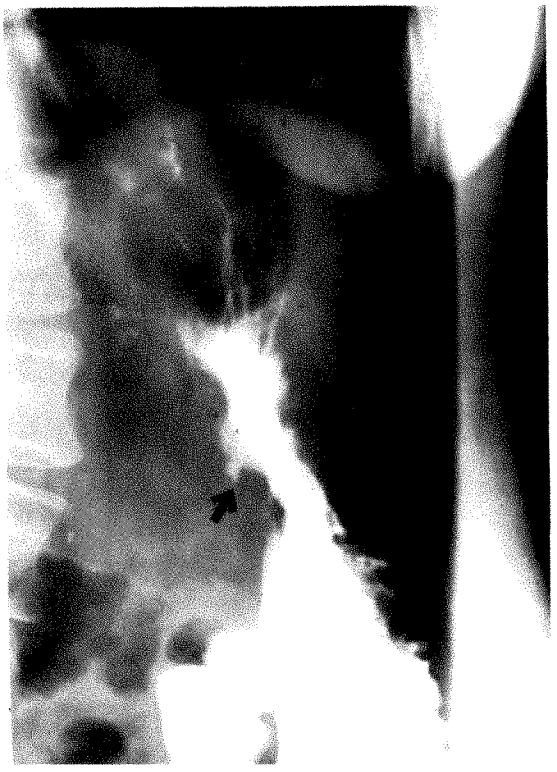


Fig. 2. Penetrating lesser curve gastric ulcer. Calcified granular and ring shadows are visible in the region of the celiac axis. There are only minute calcified plaques in the lower abdominal aorta. Calcification+.

Table IV

PERCENTAGE WITH SEVERE CALCIFICATION (++ AND +++) OF THE ABDOMINAL AORTA IN GASTRIC ULCER AND IN CARCINOMA OF THE STOMACH (MALES AND FEMALES)

Age Group (yr.)	Gastric Ulcer	Gastric Carcinoma	Difference
50–60	44.0	1.9	42.1±7.6
61–70	60.0	7.5	52.5±7.1
71+	86.4	12.5	73.9±9.3



Fig. 3. Lesser curve gastric ulcer. The linear and granular calcifications are most marked in the upper abdominal aorta. Calcification++.

surgical treatment of gastric and duodenal ulcers, local excision of the lesions was tried but was found to be ineffective. New ulcers appeared in other areas.[4]

However, recent work has changed our conception of chronic occlusive arterial disease. It has been demonstrated that a cerebral infarct is rarely due to thrombosis in the artery supplying the ischemic area. Our attention is focused on the atheromatous plaques found in the extracerebral, rather than in the intracerebral, arteries and on the cerebral circulation as a whole, rather than on the arteries immediately supplying the affected area.[20]

Similar observations have been made in hypertension, when caused by renal artery stenosis. While the atheromatous plaques are usually found at the origin of the renal artery, vascular changes distal to the renal artery stenosis have rarely been found.[1]

Larson *et al.*[17] pointed out that, "in most reported cases of obstruction of the superior mesenteric artery by atheromatous plaques, the lesions are found at the origin of that vessel. Even more distressing, obstruction is caused fairly often by extensive degenerative changes within the abdominal aorta."

It is evident from these observations that the absence of arteriosclerotic lesions in the smaller vessels distal to the atheromatous plaques in the central arteries is a common feature of chronic occlusive arterial disease.

In accordance with the above findings, it is suggested that the calcified atheromatous plaques in the upper abdominal aorta and in the celiac axis may be the cause of insufficiency or obstruction of the gastric blood supply. This would explain why the small arteries immediately supplying the area of an ulcer are often unaffected by arteriosclerosis and why local excision of an ulcer does not prevent the development of new ones.

From the evidence produced in this



Fig. 4. Large lesser curve gastric ulcer. Note the linear and granular calcifications in the upper abdominal aorta, in the celiac axis and vascular calcification near the ulcer. Calcification++.

paper, the concept is advanced that general arteriosclerosis and, in particular, its manifestation in the upper abdominal aorta and in the celiac axis play an important part in the development of gastric ulcer in the aged.

SUMMARY

1. The incidence and the severity of calcified atherosclerosis have been studied by roentgenography of the abdominal aorta in 2,185 patients, aged over 50 years, (293 patients with chronic benign gastric ulcer, 188 patients with cancer of the stomach and 1,704 control patients). It was found that patients with gastric ulcer have a significantly greater incidence and severity of calcified atherosclerosis than control cases and, in particular, than patients with carcinoma of the stomach.

2. The age and sex distribution of gastric ulcer, like coronary artery disease and cerebral vascular disease, tends to follow the age and sex distribution of calcified atherosclerosis of the abdominal aorta.

3. Independent of the extent and the severity of the calcified lesions in the abdominal aorta, calcified plaques in the upper abdominal aorta and in the celiac axis can be recognized on roentgenograms in the majority of patients with gastric ulcer.

4. It is suggested that recent work on the pathologic basis of chronic occlusive arterial disease invalidates the arguments against the arteriosclerotic pathogenesis of gastric ulcer in the aged.

5. The concept is advanced that calcified plaques in the central blood supply of the stomach may lead to arterial insufficiency

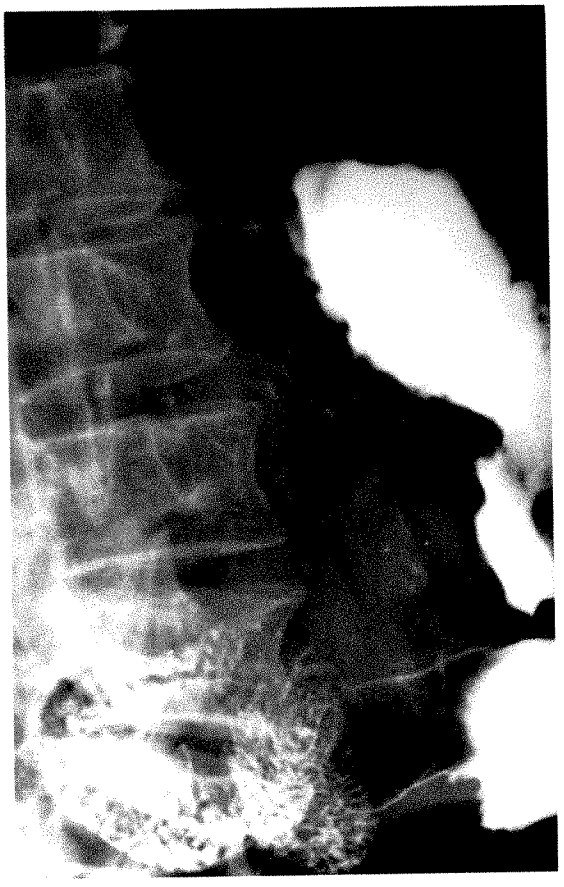


Fig. 5. Benign giant gastric ulcer. Extensive calcification of the abdominal aorta and multiple vascular calcifications along the upper lesser curve of the stomach. Calcification+++.

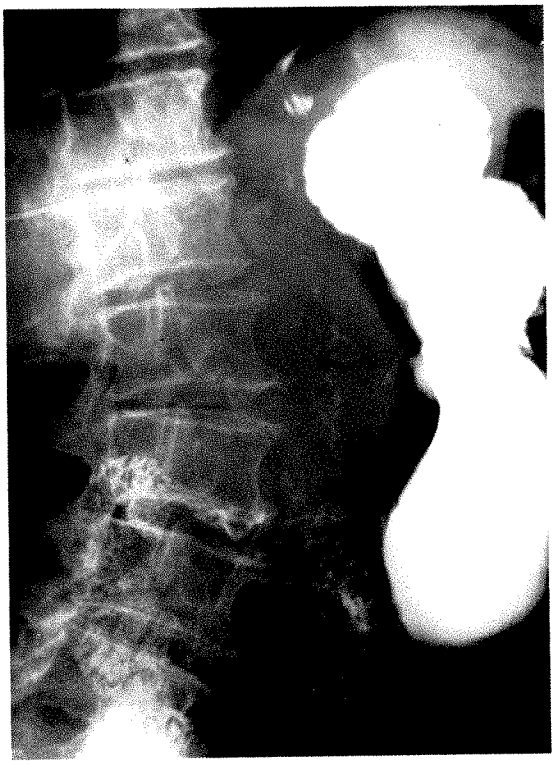


Fig. 6. Lesser curve gastric ulcer. Dense calcification of the abdominal aorta and of the celiac axis. Note the vascular calcifications near the ulcer. Calcification+++.

or obstruction and play an important part in the pathogenesis of gastric ulcer in the aged.

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PSEUDOTUMORAL GASTRIC VARICES

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THE roentgenographic recognition of gastric varices often is difficult, even when there is a history of liver disease or demonstrable esophageal varices. However, due to a rare combination of circumstances, gastric varices may occur in the absence of other diagnostic clinical findings, and thus provide an even greater problem in identification. From the point of view of surgical management, it is imperative that the true nature of the lesion be known preoperatively. A recent experience, in which the varices occurred as a gastric "tumor," prompted a review of our cases as well as of the pertinent literature in order to clarify the situation and, possibly, to provide additional diagnostic clues.

The German radiology literature contained some of the earlier descriptions of gastric varices.[3] Templeton[10] discussed the roentgenographic appearance of these lesions and stressed the difficulty in differentiating them from carcinoma in the absence of esophageal varices. Samuel[8] reviewed the venous drainage of the stomach and recognized that any obstruction of the portal or splenic vein might produce roentgenographically demonstrable gastric varices. He concluded that dilatation of the fundal veins might antedate esophageal varices, since the blood passed through them to the esophageal venous plexus, and could be the earliest evidence of variceal disease.

Evans and Delany[3] were able to find gastric varices in 65 per cent of 40 cases in which esophageal varices had been noted. However, they were apparently unable to make a diagnosis of gastric varices in the absence of other findings suggestive of portal hypertension. Smookler[9] noted the association of gastric varices with hematemesis and splenomegaly. He reported a case of a patient with alcoholic cirrhosis in which the roentgenographic appearance of the gastric varices was that of a polypoid mass suggestive of neoplasm. Surgical exploration proved the mass to be gastric varices which bled profusely. Wohl and Shore[11] described a similar episode in which the mass of gastric varices was accidentally penetrated during the course of operation and the patient did not survive. When they reviewed other cases, they found gastric varices to be present with surprising frequency in their cirrhotic patients. Also, they noted the occurrence of gastric varices in the absence of demonstrable esophageal varices and the frequent association of gastric varices with an enlarged spleen.

The venous drainage of the stomach is illustrated in Figure 1. A detailed discussion of portal hypertension is not within the scope of this report, but it might be noted, however, that Laennec's cirrhosis and obstruction of portal or splenic veins by thrombosis or tumor are common conditions causing increased portal pressure and, thereby, gastric and esophageal varices. Conversely, gastric varices have been reported in the absence of portal hypertension, and, rarely, on a congenital basis also.[9]

We reviewed all cases in the Mayo Clinic files from 1951 to 1962 in which the diagnosis of gastroesophageal varices had been confirmed surgically. This included 236 patients who had undergone some shunting procedure for portal hypertension and on whom splenoportography had been performed. Those cases in which the roentgenologist had had no prior information concerning the probability of varices were selected for further study. It was not surprising to find that the roentgenographic diagnosis of gastric varices had not been

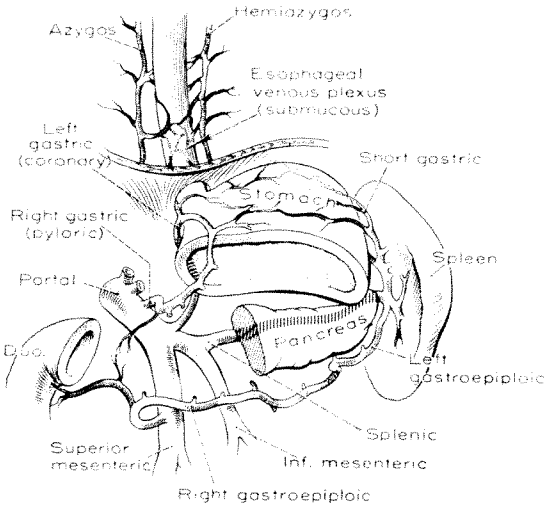


FIG. 1. Schematic drawing of the venous drainage of the stomach. (Modified, with permission from *Radiology*.[3])

made without a suggestive history available, or coexisting esophageal varices present, at the time of examination. However, we found 2 cases of isolated gastric varices with roentgenographic changes consistent with the presence of such an abnormality. These, plus a recent case, constitute this report.

REPORT OF CASES

CASE I. This 44 year old farmer first was seen here in March, 1962 after an acute episode of hematemesis. He had had melena for 2 months prior to admission.

Physical examination was nonrevealing. Neither the liver nor the spleen was palpable. The results of pertinent laboratory tests were: hemoglobin, 7.7 gm. per 100 ml. of blood; erythrocyte sedimentation rate, 6 mm. in 1 hour (Westergren); sulfobromophthalein retention, 8 per cent; direct serum bilirubin, negative; and indirect bilirubin, 1.2 mg. per 100 ml. of serum. Roentgenographic examination revealed a mass near the esophagogastric junction which suggested neoplasm, but re-examination the next day gave results reported as negative. Esophagoscopy showed an indeterminate lesion of a polypoid nature near the cardia. There appeared to be prominent folds in the esophagus; however, these were not characteristic of varices.

Because the patient still was hemorrhaging actively and requiring multiple transfusions, exploratory laparotomy was carried out. The spleen was four times larger than normal. The liver appeared to be grossly normal. Gastrotomy revealed diffuse gastritis with multiple areas of ulceration, mostly in the upper half of the stomach. The surgeon reported no evidence of portal hypertension and no evidence of a mass. Gastric biopsy confirmed the gross impression of inflammatory gastric mucosa. Because of the apparent nature of the gastric lesion, a trial of medical management seemed advisable, and no definitive surgical treatment was carried out.

The patient returned home but subsequently mild discomfort in the right upper quadrant of the abdomen developed along with icterus consistent with homologous serum hepatitis. This was followed by several more episodes of hematemesis and melena. He was re-admitted in August of 1962 with anemia, leukopenia, and thrombocytopenia. Roentgenograms made at that time were reported negative. The patient's problem now was thought to be of a chemotoxic nature or, possibly, idiopathic hypersplenism. Transfusions of blood were given again and he was advised to avoid sprays and chemicals. Less than 2 weeks later he returned with recurrent massive hematemesis and melena. The spleen was again palpable. After several transfusions, a third roentgenographic barium meal study revealed an unequivocal polypoid filling defect in the cardia (Fig. 2, *a* and *b*).

Because of continued bleeding, emergency exploration was carried out. Obvious cirrhosis, probably posthepatitic, was now seen in the liver and this was confirmed by biopsy. The spleen was four to five times normal size. Portal pressures from the superior mesenteric vein ranged from 9 to 10 cm. of saline (within normal limits).

A soft "tumor," covered with mucosa, was seen on opening the upper third of the stomach. This was found to consist of a large mass of venous varicosities near the esophagogastric junction associated with several distended short gastric veins. Since portal hypertension was absent, partial gastrectomy, splenectomy, and pyloroplasty were performed. The postoperative course was relatively uneventful, and the patient was dismissed on the twelfth postoperative day. When last seen, 5 months later, he had experienced no further bleeding.

CASE II. This 52 year old man had noted several recurrent episodes of hematemesis and

melena starting 3 years prior to admission. He had a past history of hypertension and coronary infarction with congestive failure.

Physical examination revealed the liver to be palpable three fingerbreadths below the costal margin. The spleen was questionably palpable. The pertinent laboratory data were: hemoglobin, 14.6 gm. per 100 ml. of blood; erythrocyte sedimentation rate, 37 mm. in 1 hour; sulfobromophthalein retention, 10 per cent; direct serum bilirubin, negative; indirect serum bilirubin, 0.8 mg. per 100 ml.; thymol turbidity, 4+; zinc sulphate, 19; cephalin flocculation, 2+; albumin-globulin ratio, 4.3/3.3; and total protein, 7.1 gm. per 100 ml. of serum. These findings were suggestive, but not diagnostic, of hepatic cirrhosis. Examination of the stomach revealed a small, but smooth and well defined, polypoid filling defect in the cardiac end of the stomach, thought to represent prolapse of the esophageal mucosa (Fig. 3, *a* and *b*). Esophagoscopy showed no evidence of varices. At gastroscopy, a mass the size of a walnut was seen arising from the cardia and apparently was covered with normal mucosa. It was thought to be an intramural lesion, possibly a leiomyoma.

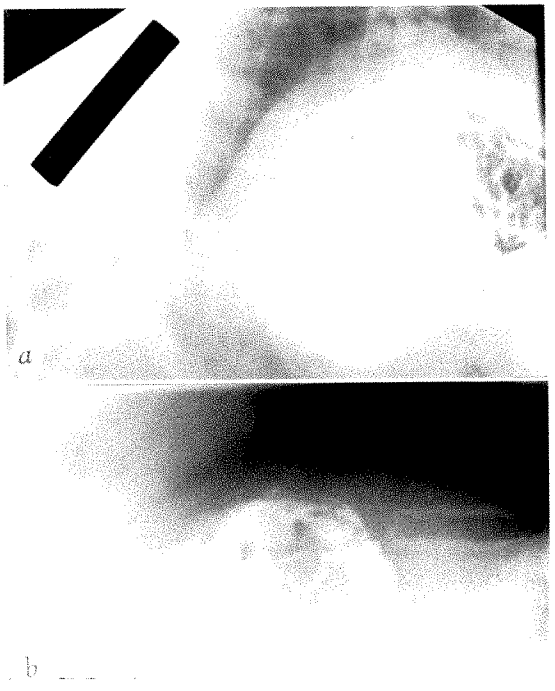


Fig. 2. Case 1. Patient was a 44 year old man with hematemesis and splenomegaly. (*a*) Barium meal showed "mass" near the esophagogastric junction, later proved to be gastric varices. (*b*) Spot roentgenogram of the cardia, with the patient lying on his right side, shows smooth-walled polypoid nature of the varices, easily mistaken for a neoplasm.

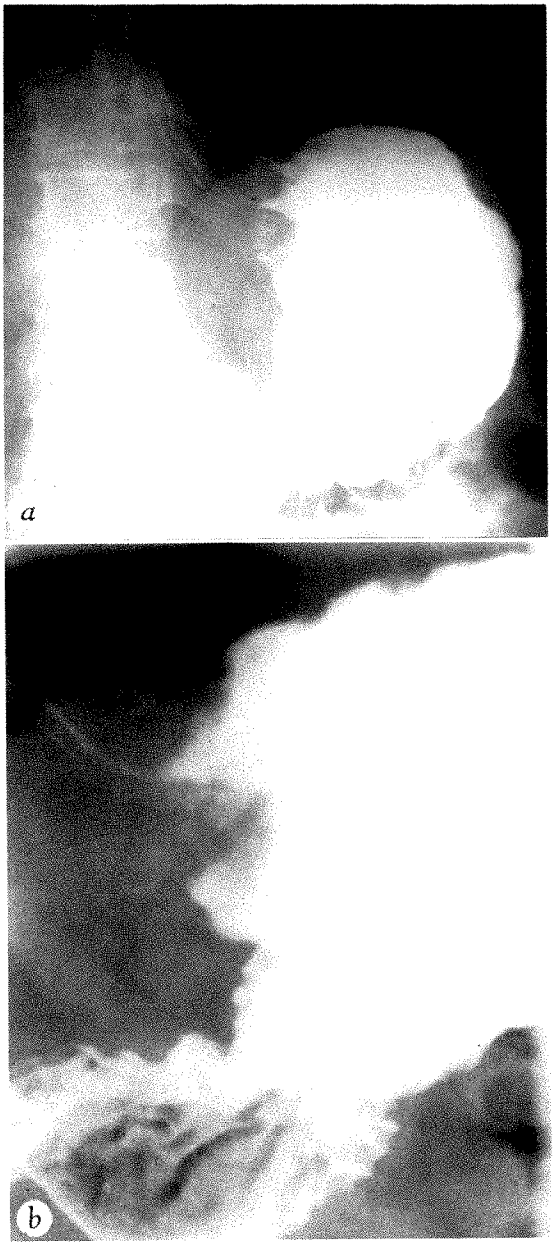


Fig. 3. Case 11. Patient was a 52 year old man with hematemesis and hepatosplenomegaly. (*a*) Roentgenogram reveals a filling defect in the region of the esophagogastric junction. (*b*) Spot roentgenogram shows irregular deformity, suggestive of malignant lesion, but found to be gastric varices at operation.

Surgical exploration revealed hepatic cirrhosis, which was confirmed by biopsy. The portal pressure was 25.0 cm. of saline (more

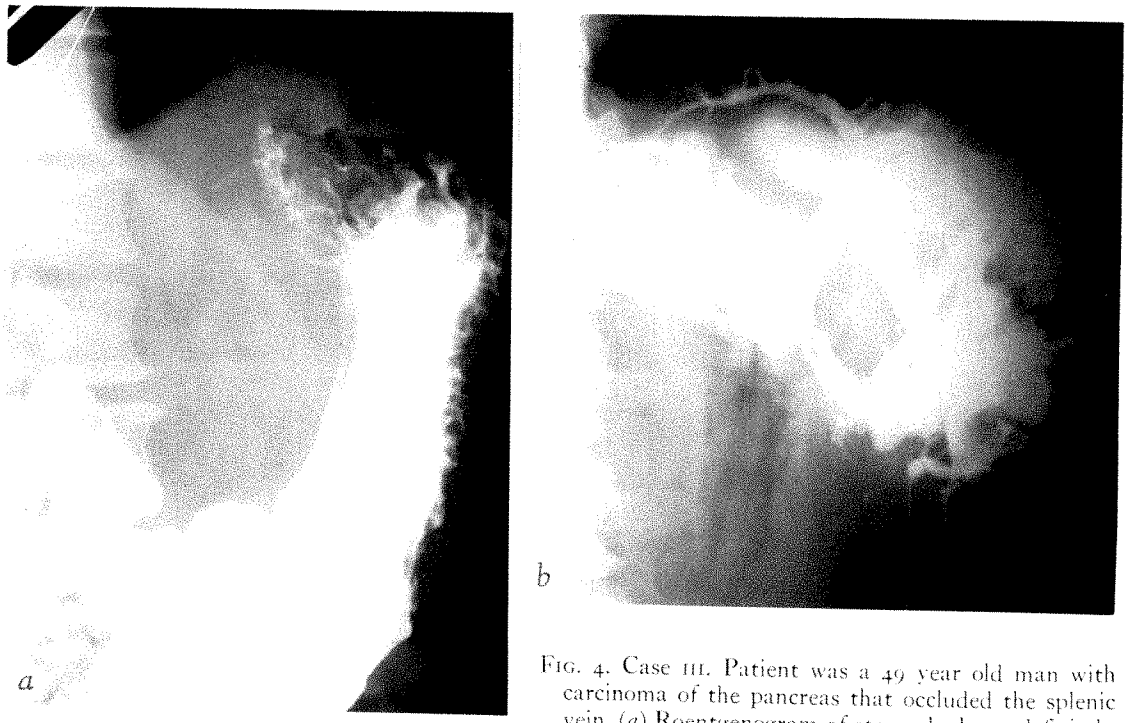


FIG. 4. Case III. Patient was a 49 year old man with carcinoma of the pancreas that occluded the splenic vein. (*a*) Roentgenogram of stomach shows definitely exaggerated mucosal folds confined to the cardia. (*b*) Preoperative splenoportogram reveals complete obstruction of the splenic vein. Large, dilated, short, gastric veins form collateral channels and join with a nest of tortuous subserosal gastric varices in the cardia.

than normal and in the range of portal hypertension ordinarily associated with gastrointestinal hemorrhage). Exploration of the stomach was negative for any masses or other lesions. There were, however, several huge venous communications between the spleen and the stomach. In the absence of suitable veins for shunting purposes, splenectomy was carried out. The patient did well postoperatively, but died 1 year later of a heart attack, without recurrence of bleeding.

CASE III. A 49 year old man was admitted because of pain in the left upper quadrant of the abdomen and weight loss during the preceding 5 months. There had been one episode of hematemesis in the past.

Physical examination revealed a palpable spleen. The pertinent laboratory data were: hemoglobin, 12.8 gm. per 100 ml. of blood; erythrocyte sedimentation rate, 34 mm. in 1 hour; sulfobromophthalein retention, 0 per cent in 30 minutes; direct serum bilirubin, negative; indirect serum bilirubin, 0.46 mg. per 100 ml.; total serum protein, 6.25 gm.; and albumin-globulin ratio, 4.60/1.65. Esophagoscopic examination and roentgenographic examination of the stomach were reported as negative, but, in retrospect, the latter showed an exaggerated, thick, tortuous mucosal pattern in the region of the cardia (Fig. 4*a*). Because of the suspicion of liver disease in this case, preliminary splenoportography was performed in the operating room and it showed complete obstruction of the splenic vein (Fig. 4*b*). At surgical exploration, an islet cell carcinoma of the tail of the pancreas was found which had obstructed the splenic vein and caused great engorgement of the short gastric veins. The gastroepiploic venous system was similarly dilated and tortuous and large subserosal gastric varices were present. The tumor was not resectable. However, the spleen was removed in an attempt to decrease the risk of further hemorrhage from the gastric varices. The patient recovered from the operation, but experienced a gradual deterioration and died 1 year later from the malignant neoplasm.

COMMENT

We have attempted to draw certain conclusions from our limited experience with this unusual roentgenographic finding.

As is well appreciated among roentgenologists, the examination of the upper portion of the stomach and the region of the esophagogastric junction is seriously limited. In addition, the accuracy of detecting coexisting esophageal varices, if they are present, varies from 15 to 68 per cent.[1,2] Among a group of 236 patients treated surgically at the Mayo Clinic for bleeding gastroesophageal varices, roentgenographic evidence was apparent in 69 per cent of patients with cirrhosis and 51 per cent of those with extrahepatic portal obstruction.[1,7]

To be sure, the accuracy of roentgenographic examination generally is enhanced when there is clinical suspicion of varices. On the other hand, while clinical evaluation usually is helpful, at times it may be misleading. Also, as with esophageal varices, gastric varices may be present but not demonstrable roentgenographically.

By the same token, the roentgenologist should understand the limitations of clinical diagnostic procedures commonly employed, as well as the difficulty often encountered in elucidating the cause of acute bleeding in the upper part of the gastrointestinal tract. In any event, it becomes apparent that in the small group of patients in which there is failure to suspect varices clinically, the astute roentgenologist may be of real assistance if he keeps certain salient features of the problem in mind. In each of our cases, there was a history of hematemesis usually associated with melena. In spite of this, a history of alcoholism, pre-existing hepatitis, or other symptoms suggesting involvement of the portal system could not be elicited. Since physical examination revealed an enlarged spleen in our 3 cases, we think that this is a highly suggestive finding. Laboratory studies, including tests of liver function, were inconclusive in all. Likewise, endoscopy was not of diagnostic value in this particular group.

Roentgenologic examination in these 3 cases showed a well-defined filling defect near the esophagogastric junction in 2, and abnormally thick, tortuous mucosal folds in the region of the cardia in the third. Obviously, these findings bring to mind more common entities such as polypoid gastric carcinoma, leiomyoma, lymphoma, or Menetrier's disease;[4] but, the presence of such a filling defect or exaggerated folds in a patient with a history of hematemesis and with splenomegaly should alert the examiner to the possibility of gastric varices. In such a situation, the fluoroscopist himself might palpate for an enlarged spleen when this information is not readily available.

Should gastric varices be considered a likely cause of bleeding, splenoportography logically is the next step, and might enable a precise diagnosis to be made prior to operation. If surgical intervention then is deemed advisable, it can be planned appropriately. From the surgeon's point of view, the operative exposure and positions required for the control of bleeding from gastroesophageal varices are quite specific and must be selected on the basis of preoperative information. The usual incisions employed for abdominal exploration, although adequate to establish the diagnosis of undetermined gastrointestinal bleeding in most instances, must be modified or closed to accomplish the surgical treatment of varices. Likewise, many so-called blind resections for hemorrhage in the upper part of the gastrointestinal tract could be avoided by more precise preoperative diagnosis.

Percutaneous splenoportography, supplemented with measurements of portal pressure, performed in the operating room prior to laparotomy is important in verifying the presence of varices and defining the exact nature of the portal obstruction as either intra- or extrahepatic.[6] In addition, it is an aid in planning a splenorenal or portocaval shunt, or an appropriate resective procedure.[5] Finally, having roentgenographic facilities in the operating room and a cassette beneath patients subjected to surgical exploration for gastrointestinal bleeding may prove advantageous in this situation, as well as in the occasional case

with unexpected operative findings, by lending greater diagnostic flexibility in a difficult situation.

SUMMARY

An apparent polypoid filling defect or exaggerated mucosal folds in the proximal portion of the stomach, accompanied by hematemesis and splenomegaly, should suggest the presence of gastric varices. Although infrequent, the lesion must be differentiated from polypoid carcinoma and other less common benign conditions occurring in this location. Splenoportography is a valuable diagnostic adjunct and enables the surgeon to plan a more appropriate operative approach.

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PHARYNGEAL AND ESOPHAGEAL PERFORATIONS DURING INSTRUMENTATION*

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WITH more widespread use of instruments designed to pass through the esophageal lumen for purposes of visualization, biopsy or dilatation, iatrogenic perforation of the esophagus and contiguous structures is being seen more and more often. It is estimated that close to 80 per cent of all esophageal perforations are due to instrumentation.[3] It is the purpose of this presentation to review the clinical findings, the roentgenographic appearance and the prognosis of esophageal perforations using selected cases from our files as illustrative examples.

CLINICAL CONSIDERATIONS

The frequency with which the esophagus and the hypopharynx are perforated during instrumentation depends upon several factors: (1) The experience of the operator is of inestimable importance. The more experience he has, the less often perforations occur, both because of refinements in his technique and in his selection of patients. (2) The presence of pre-existing mucosal disease increases the risk of perforation, for this is the strongest barrier in the esophagus. Consequently, attempts at dilatation of the diseased esophagus in which one purposefully ruptures the mucosa carries a greater risk of perforation than does esophagoscopy *per se*. (3) Large cervical osteophytes predispose to perforations as does (4) any anomaly which further encroaches upon the areas of natural narrowing of the esophagus, *i.e.*, at the cricopharyngeus, at the arch of the aorta and at the entrance of the esophagus through the diaphragm. (5) Of considerable importance in procedures done under local anesthesia is the extremely tense and uncooperative patient. The wise operator will either do the procedure under general anesthesia or cancel it altogether under this circumstance. While no procedure on a patient is to be taken lightly as illustrated in the cases below, esophagoscopy and gastroscopy are relatively safe procedures in experienced hands when compared with the information gained. Palmer and Wirts[4] reviewed 40,540 procedures by questionnaire and found that the mortality from esophagoscopy was only 0.059 per cent while the accident rate was 0.25 per cent. Gastroscopy carried a mortality of only 0.014 per cent with an accident rate of 0.079 per cent in 267,175 procedures. Anesthetic reactions constituted 18 per cent of the gastroscopic accidents and 6 per cent of the esophagoscopic accidents. The morbidity of esophagoscopy and gastroscopy due largely to perforations is generally accepted as less than 2 per 1,000 procedures done. Paradoxically, the relative safety of esophageal instrumentation leads to more procedures being done on more patients with an increase in the actual number of perforations but with little or no change in mortality rates.

Mortality depends upon the site of perforation, whether or not the pleural spaces are involved, the rapidity with which the perforation is recognized and repaired and, of course, the condition of the patient at the time of reparative surgery. Perforations of the cervical esophagus and hypopharynx usually occur at or just above the cricopharyngeus muscle, called the "Gate of Tears."[2] Such a perforation may result from penetration of one of the lateral pyriform sinuses but more commonly occurs in the posterior wall in the weak area produced by the "sling" formation of the crico-

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pharyngeus muscle. Perforation in this area may also result when pressure by the instrument on prominent cervical osteophytes produces a laceration allowing the escape of air and contaminated fluids into the loose connective tissue of the prevertebral "space." Such a perforation may be detected later than other types of perforation but carries a better prognosis. Perforations in the cervical esophagus have a mortality of about 15 per cent or less.[3]

Mortality of perforations occurring in the mid or lower esophagus varies between 25 per cent and 35 per cent when the pleural spaces are involved. If the pleural cavity is not involved, the mortality is similar to that of cervical perforations. The greater mortality in the thorax is probably due to greater inaccessibility to reparative surgery and to the greater contamination of the mediastinum and pleura by reflux of acid gastric juice. The prevertebral area is a potential space presenting no barriers to widespread dissemination of air, bacteria, fluid and contrast material. Any of these foreign substances can easily present themselves in the neck as well as throughout the mediastinum. Perforations in these areas thus allow massive pneumomediastinum and pneumothorax with their associated complications such as cardiac arrythmias. The authors have had no difficulty with barium in the mediastinum although such contamination is to be avoided. A similar experience is recorded by Heald.[1] Case L.T. described below has been followed for over 1 year after massive contamination of the mediastinum by barium and no residuals have been noted.

The primary symptom of pharyngeal or esophageal perforation in the cervical area consists of a "sticking" sensation low in the throat which is aggrevated by attempts to swallow. This may be relatively mild when first noted, especially if due to maceration of the lumen's wall, which allows only slow but progressive contamination of the prevertebral space. When the procedure is done under local anesthesia subcutaneous emphysema may be noted before pain develops. Air dissects easily down through the loose tissue of the prevertebral space of the cervical area into the mediastinum, and chest roentgenograms may reveal the "Minnigerode sign" or bubbles of gas in the periesophageal tissues. Pain may play such a small role initially that on rare occasions the physician is unaware of the potentially fatal complication of mediastinitis or abscess until fever, leukocytosis or progressive dysphagia is noted 24 hours or more after the procedure.

The outstanding clinical manifestation of perforations of the intrathoracic esophagus is excruciating, substernal pain with radiation through to the interscapular area and, if the pleural spaces are involved, laterally. Characteristically, this pain cannot be relieved with narcotics. It is remarkable that perforation is seldom recognized by the operator at the time the wall is disrupted.

ROENTGENOLOGIC CONSIDERATIONS

The factual diagnosis of esophageal perforation rests almost solely on roentgenologic investigation.

The recognition of a pharyngeal or esophageal perforation reontgenologically can be proven by certain primary evidence, and can be strongly suspected by certain secondary evidence.

The primary evidence of pharyngeal or esophageal perforation is the identification of contrast material in an extraluminal location, following its administration orally. This material may be adjacent to or at some distance from these structures, and may include the cervical area, the mediastinum, the pericardial space, one or both pleural spaces, the peritoneal cavity, and the retroperitoneal space.

The secondary evidence of perforation is the identification of abnormal air and fluid collections in the cervical area, the mediastinum, the pericardial space, one or both pleural spaces, the peritoneal cavity and the retroperitoneal space.

Ideally, the examination should be done in the radiology department, rather than with a mobile roentgenographic unit. Ade-

quate examination is vitally important, and should include fluoroscopy (preferably with image intensification) at the time of oral administration of a water soluble contrast medium, followed by appropriate roentgenograms of the suspected areas of involvement (cervical, chest, upper abdomen).

ILLUSTRATIVE CASES

Case I. R.C. Female, aged 81 years. This elderly female underwent an elective cholecystectomy. Following a difficult intratracheal intubation, the operation was carried out with ease. After the surgical procedure, subcutaneous emphysema of the neck was noted by palpation. A roentgenographic chest examination with the mobile unit in the recovery room revealed the presence of a moderate amount of air in the mediastinum and cervical area (Fig. 1). She responded well to medical management, and subsequent examinations during the next few days showed regression of the emphysema.

Comment. Such cases as this are thought to be pharyngeal (or esophageal) tears resulting from the hard surface of the anesthesia instrument rubbing against the vertebrae of the hyperextended cervical spine, particularly when there are large osteophytes on the anterior borders of the bodies. (A lateral chest roentgenogram on this patient made prior to surgery showed such osteophytes on several of the lower cervical vertebrae.)

Case II. L.T. Female, aged 58 years. This patient gave a history of vague upper abdominal complaints. An upper gastrointestinal series disclosed a mild constriction of the gastric antrum. Because of this, the Hirschowitz fiberduodenoscope was introduced in a sitting position. A local anesthetic to the larynx was employed. The usual resistance to passage of the instrument was felt at the level of the cricopharyngeus muscle, but the instrument then advanced without difficulty to a level of 36 cm. from the incisor teeth. At this time the patient experienced a very severe substernal pain which radiated through to the back. This pain was unrelieved by 75 mg. of demerol. She stated that this was similar to the distress she had been having, although in retrospect this was doubtful.

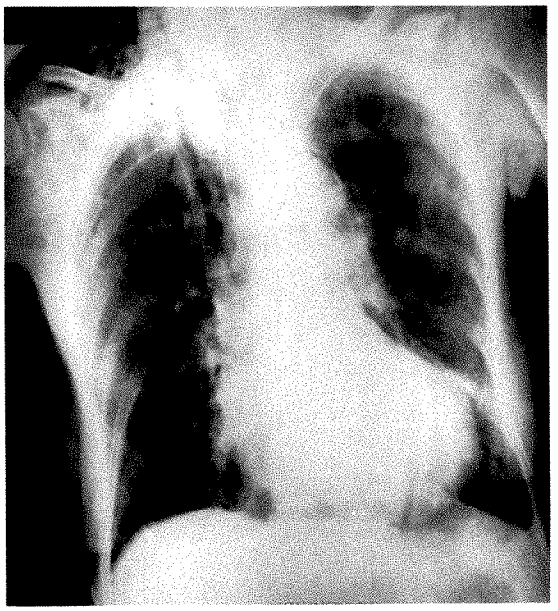


Fig. 1. Case 1. Frontal chest roentgenogram shows moderate amounts of air within the mediastinum and cervical area. The lateral border of most of the thoracic aorta is outlined by air. The ribbon-like opacity in the upper mediastinum on the right is thought to be the superior vena cava outlined by air within the right lung and the mediastinum.

Roentgenologic examination of the esophagus was done immediately. Inadvertently, a barium sulphate preparation was used, rather than a water solub'e medium. A posterior wall perforation was disclosed at the C6–7 level (Fig. 2, *A* and *B*). The extravasated contrast material, at the end of the examination, had extended downward into the mediastinum to the level of D8 (Fig. 2*C*). A surgical repair was begun within 2 hours of the perforation. The cervical area and upper mediastinum were suctioned, and the rent in the esophagus closed. Her postoperative course was uneventful except for the development of an esophagocutaneous fistula, which closed spontaneously.

One mounth later, examination of the esophagus disclosed further extension of the residual contrast medium downward in the mediastinum, with some of it around the esophagus, but the greater bulk more posteriorly around the aorta (Fig. 2, *D* and *E*). Several subsequent examinations have shown no essential change in the position or amount of the medium. There has been no evidence of ill effect from this foreign substance after more than a year.

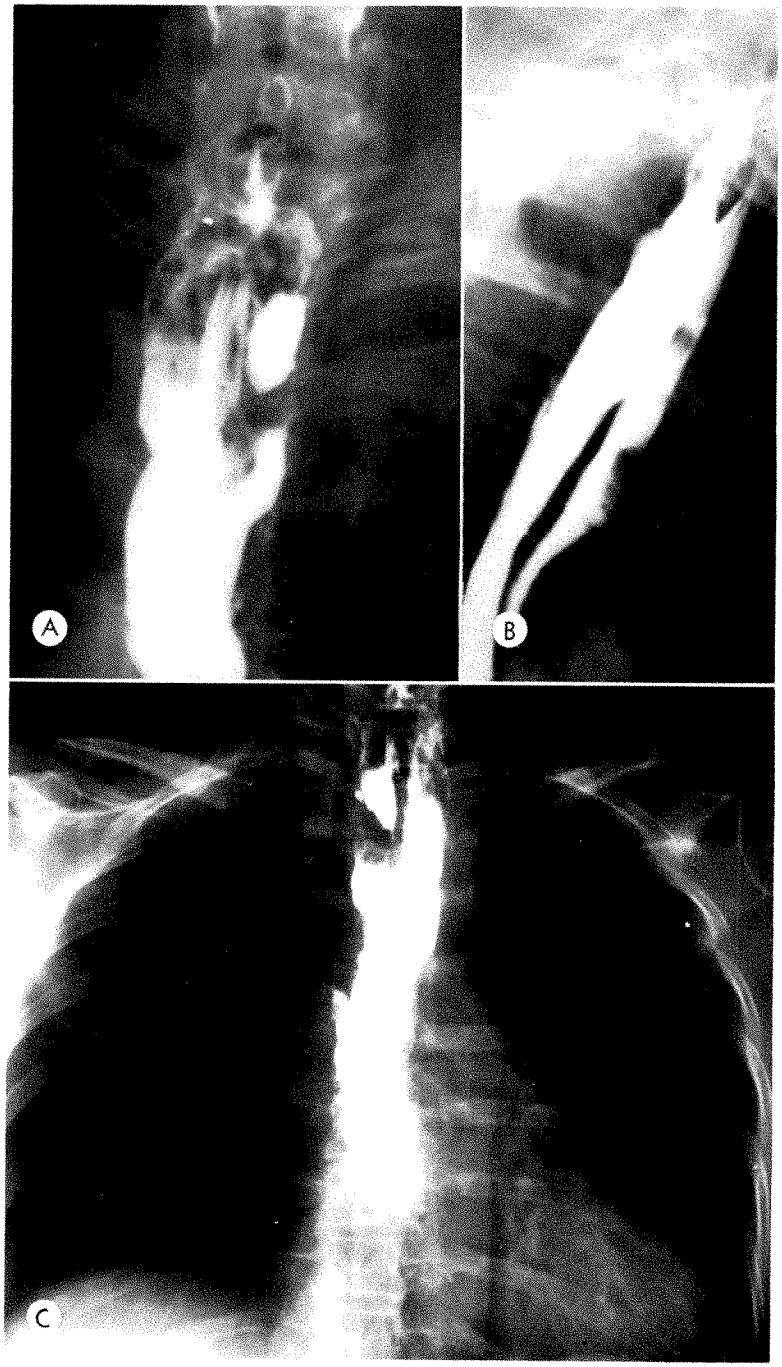
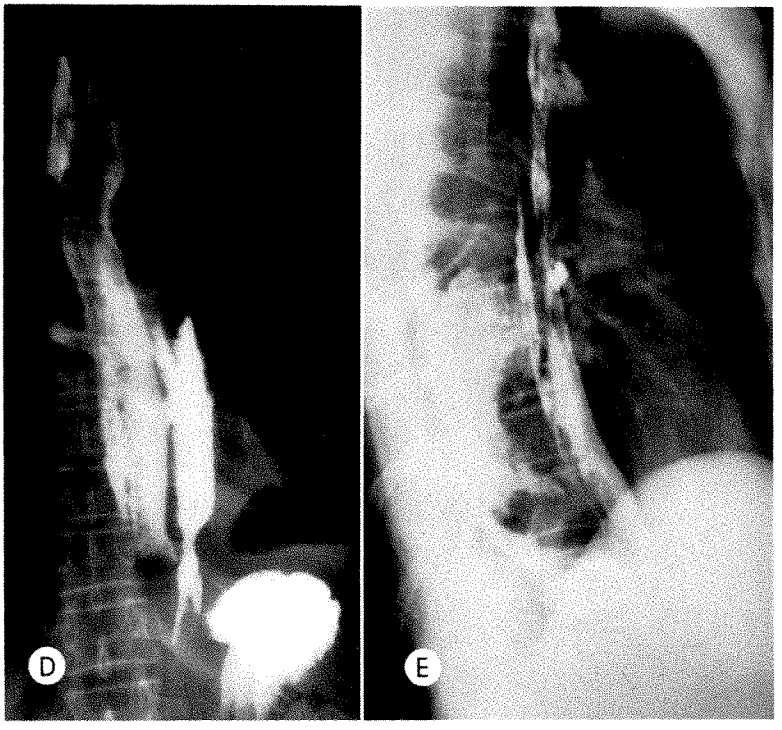


Fig. 2. Case II. Frontal (*A*) and oblique (*B*) roentgenograms of the lower cervical area and upper mediastinum, made during the fluoroscopic examination, show two tracts of barium, the shorter anterior tract being within the esophagus, the longer posterior tract being in the prevertebral space. The site of perforation in the lower cervical area is outlined by the contrast material in the oblique view. (*C*) Frontal roentgenogram of the mediastinum at the end of the examination reveals downward extension of the opaque material to the approximate level of the fiberscope's penetration.



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Fig. 2. Case II. (*D* and *E*) Appearance of the mediastinum 1 month later, demonstrating the location of the barium around the esophagus and aorta. The lower half of the esophagus is shown to contain opaque material in these views.

Comment. This patient is interesting from several aspects. She is the sixth reported case of perforation with the flexible fiberscope. The perforation in this presumably healthy esophagus occurred at one of the fixed points where perforations so frequently occur. The fiberscope passed with such ease through the mediastinum that the operator was unaware of it being outside of the esophagus. The local anesthetic which was used in the pharnyx probably masked the clinical findings to some extent for both the patient and the operator.

Case III. O.J.H. Female, aged 69 years. This patient gave a 2 year history of dysphagia, vomiting and a 20 pound weight loss. The esophageal studies revealed a moderate constriction in the middle third. Esophagoscopy revealed the lesion, and a biopsy done during the examination was later reported as "hyperkeratosis and leukoplakia." The operator did not recognize a perforation at the time of the procedure. Postoperatively, however, the patient complained of marked exacerbation of her preoperative pain upon swallowing. An immediate roentgenographic chest examination with the mobile unit was negative. A contrast esophageal examination with lipiodol the next day disclosed no leakage.

Seven days after endoscopy, because of continued symptoms, another esophageal examination was done. This disclosed a large hole in the esophagus at the site of the previously disclosed lesion, continuing into a walled off pocket in the lower posterior mediastinum (Fig. 3 *A*). Gastrostomy was performed, and massive antibiotics were instituted. Her condition improved and subsequently she was discharged from the hospital.

Ten weeks later the patient was re-admitted for further treatment. An esophageal roentgenologic examination again disclosed the lesion, but there was no fistulous communication identified. Endoscopy and biopsy were done, the latter again being reported "hyperkeratosis and leukoplakia." With considerable difficulty, the operator positioned a Souttar tube. Again the patient experienced an exacerbation of her symptoms, and a roentgenographic examina-



Fig. 3. Case III. (*A*) Oblique roentgenogram of the chest discloses perforation of the esophagus in its middle third, with a localized abscess in the posterior mediastinum which is filled with contrast material (lipiodol). The lower esophagus is displaced anteriorly by the abscess pocket. (*B*) Reperforation of the esophagus during placement of a Souttar tube (10 weeks after the initial perforation). The phrenoesophageal ligaments evidently have been ruptured, allowing air and the contrast material to penetrate downward below the level of the diaphragm.

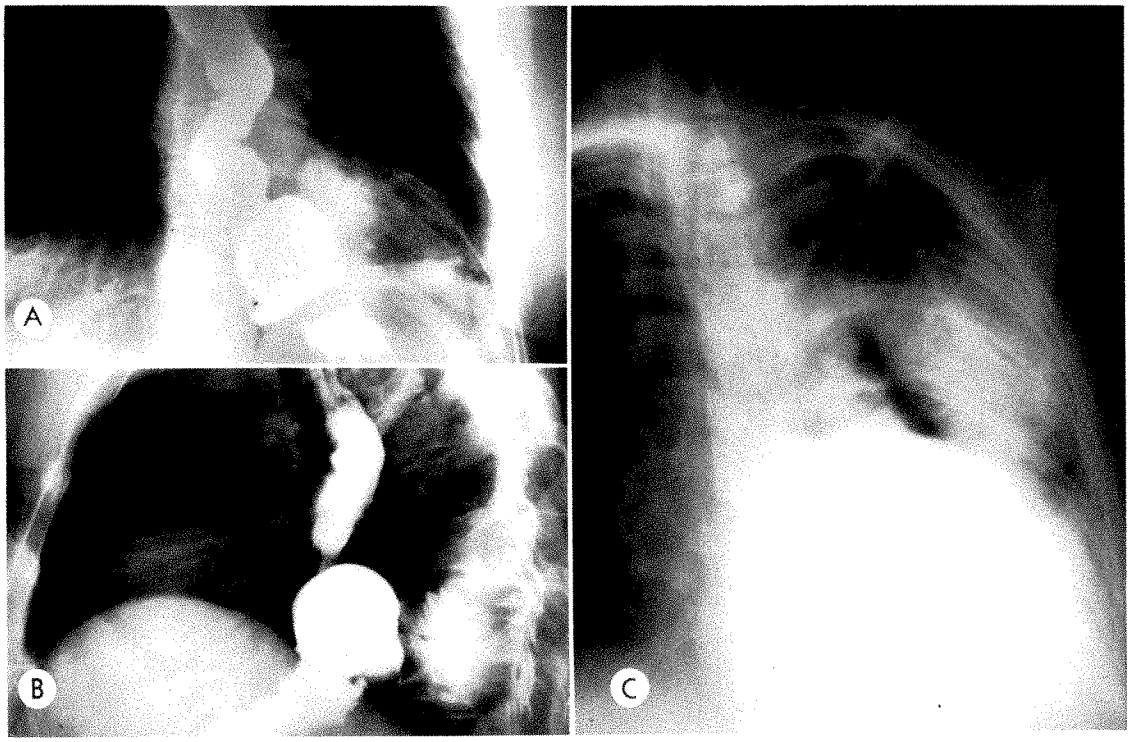


Fig. 4. Case iv. Roentgenograms of the esophagus in frontal (*A*) and lateral (*B*) views, reveal extravasation of the contrast medium from its lower end, with extension into the posterior mediastinum and left pleural space. The dilated segment immediately above the left hemidiaphragm, resembling a hiatal hernia, proved to be a dilated lower esophagus. (*C*) Chest roentgenogram made with the mobile unit 3 hours later discloses progressive changes in the mediastinum and left pleural space as a result of the esophageal perforation.

tion of the chest with the mobile unit disclosed air in the mediastinum, peritoneal cavity and retroperitoneal space. A contrast esophageal study showed that the medium which passed from the caudad end of the Souttar tube entered the upper abdomen—not the stomach (Fig. 3 *B*). This indicated that the tube, when being positioned, had penetrated the esophageal wall (presumably at the same site as the previous perforation) and was lying partly in the old walled off pocket. Further, the lower end of the tube evidently had also penetrated into the upper abdomen, accounting for the free air and opaque medium seen below the level of the diaphragm.

Comment. The esophagus in this patient was first perforated by biopsy, and then reperforated (presumably at the same site) by the introduction of a Souttar tube. The case further exemplifies involvement of the upper abdomen as well as the mediastinum when the phrenoesophageal ligaments are ruptured.

Case iv. M.P.D. Female, aged 64 years. In 1951, because of dysphagia, an esophageal examination was done and reported as "megaesophagus, probably due to cardiospasm." In 1953 a Hellar myotomy was accomplished without undue incident. For the next 7 years she was relatively asymptomatic. In 1960, she had a return of her symptoms of dysphagia. An esophagram revealed a stricture of the lower esophagus. Following this, an esophagoscopy was done under general anesthesia. A lumen finder was first used, and was thought by the operator to have entered the stomach. The endoscope, however, would not pass a stricture in the lower esophagus. No mention of perforation was made in the operative report. Upon awakening in the recovery room, the patient began complaining of very severe lower substernal pain with radiation to the back. A

roentgenographic examination in the radiologic department showed an extravasation of the medium into the lower posterior mediastinum and into the left pleural space (Fig. 4, *A* and *B*).

Three hours later, while in the recovery room, a chest roentgenogram made with the mobile unit disclosed progressive changes in the mediastinum and left chest (Fig. 4 *C*). Seven hours after the esophagoscopy, the patient underwent surgery and the perforation was closed. Recovery was uneventful.

Comment. As was true here, and as has been mentioned repeatedly, the operator was unaware that he had perforated the esophagus at the time of the procedure. However, upon reacting from the general anesthetic, the patient's symptomatology was characteristic, and proper procedures were instituted.

In this case, it is noteworthy that there was no visible subcutaneous air and only minimal mediastinal air.

Case v. G.E.B. Male, aged 54 years. This patient had a long history of reflux esophagitis. In 1960, a hiatal hernia had been repaired with poor relief of symptoms. In 1962, an esophagoscopy was performed under general anesthesia. During the procedure, a perforation was induced but not immediately suspected. A biopsy at the time was later reported as "normal lung tissue." After the termination of the esophagoscopy, the patient was cyanotic. A right tension pneumothorax was demonstrated by thoracentesis and by roentgenographic chest examination with the mobile unit (Fig. 5 *A*). There was a short period of cardiac standstill which was reversed by external massage.

When the patient's condition was stabilized, the esophagus was examined roentgenologically by contrast medium. A perforation was identified in its lower third. The medium extravasated into the posterior mediastinum and the right pleural space (Fig. 5 *B*). The perforation was closed surgically within 6 hours. This was followed by prolonged convalescence.

Comment. This case illustrates how easily a diseased eosphagus may be perforated and not suspected. It also illustrates the dramatic suddenness with which a patient's condi-

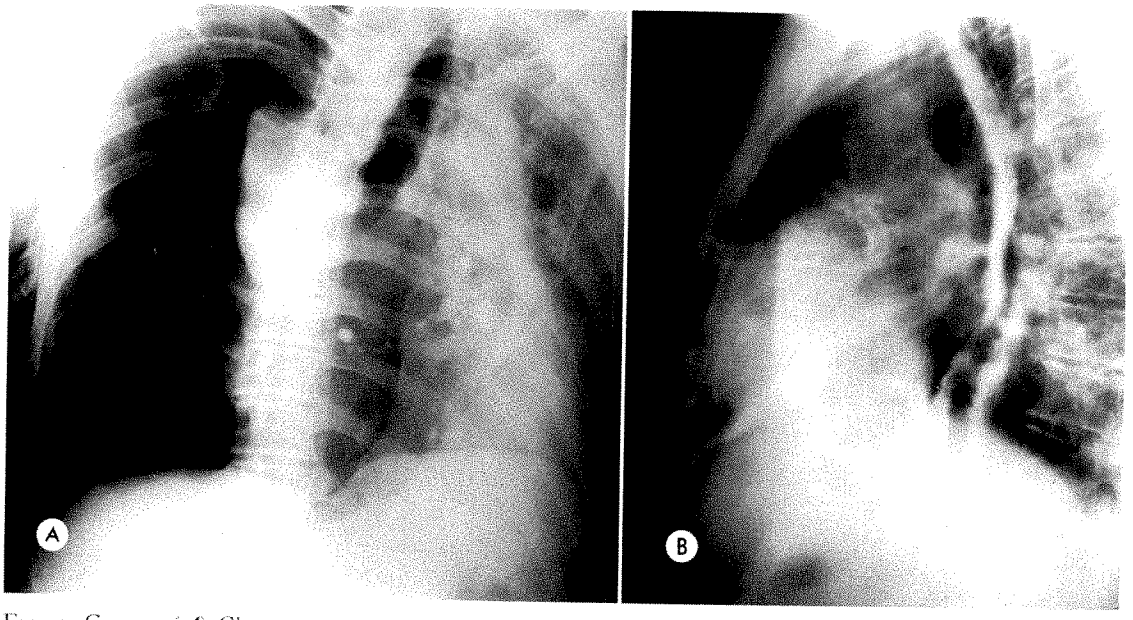


Fig. 5. Case v. (*A*) Chest roentgenogram made with the mobile unit in slightly oblique position discloses marked collapse of the right lung, following esophagoscopy and gastroscopy with biopsy presumably of the esophagus. The subcutaneous emphysema in the right chest wall is the result of the thoracentesis for relief of the tension pneumothorax. (*B*) Contrast esophageal roentgenogram, following relief of the tension pneumothorax, shows the level of the perforation, with extravasation of medium from the esophagus into the mediastinum and lower right hemithorax.

tion may be reversed by a tension pneumothorax.

SUMMARY

The problems encountered in perforation of the esophagus by instrumentation are briefly reviewed.

Five cases from a much larger series are presented to illustrate the major roentgenologic findings.

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STRANGULATED RICHTER'S HERNIA OF THE STOMACH

A CASE REPORT

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A DIAPHRAGMATIC hernia may present a myriad of bizarre roentgen manifestations. Once considered, appropriate roentgenographic studies will generally verify the presence of a hernia and identify the structures involved.

This is, to our knowledge, the first report

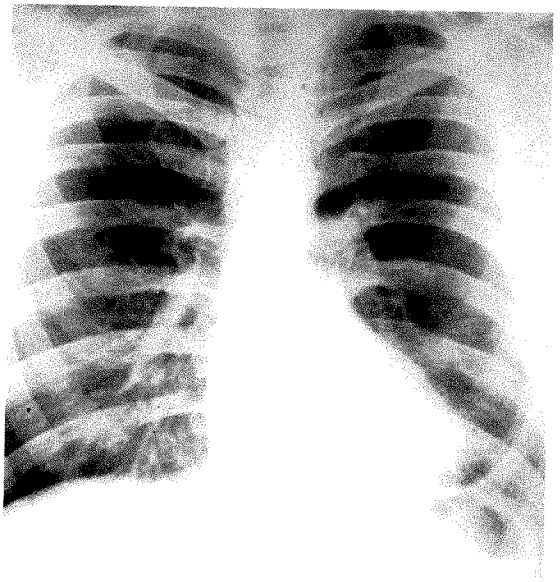


Fig. 1. Chest roentgenogram taken during an asymptomatic interval between the thoracic injury and the current admission. The healed rib fractures on the left can be seen.

of a case of an unusual type of herniation of the stomach. In spite of strong clinical suspicion, and adequate roentgenologic examination, the diagnosis was not made for some time. The delay resulted in serious, though fortunately not fatal, complications.

REPORT OF A CASE

J.M., an apparently healthy, 27 year old white male dental student, while at a social gathering, ingested moderate quantities of malt liquor. Later in the evening he vomited. The following morning he awoke with severe, constant pain in the left upper quadrant. His physician admitted him to the hospital for observation.

Three years prior to the present admission the patient was in an automobile accident and suffered fractured ribs with a left pneumothorax. The past history was otherwise unremarkable. A chest roentgenogram following recovery from the injury showed residual blurring of the left hemidiaphragm (Fig. 1).

The chest roentgenogram on admission revealed a considerable amount of fluid in the left chest obliterating the diaphragm. A fluid level was noted in the mid-portion of the left

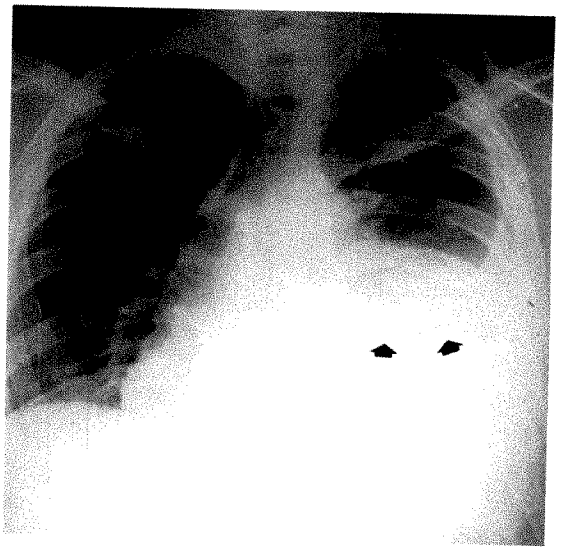


Fig. 2. Chest roentgenogram on admission showing opacification of the lower left hemithorax, mediastinal shift to the right, and the fluid level superimposed over the posterior portion of the eighth left rib (arrows).

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hemithorax. There was a shift of the mediastinal structures to the right (Fig. 2). A transdiaphragmatic herniation of a portion of the gastrointestinal tract was considered the most likely diagnosis and a barium meal study was expected to give prompt verification. The contrast material passed readily through the esophagus, stomach and entire bowel. An upright lateral chest roentgenogram showed the relationship of the fluid level in the chest to the esophagus and the stomach (Fig. 3). Despite manipulation and the Trendelenburg positioning, an abnormality in the site of a hollow viscus could not be demonstrated (Fig. 4).

Several thoracenteses were subsequently performed. Initially a thin, bloody, serous fluid was obtained, later changing to a syrupy, dark blood. Chest roentgenograms following thoracentesis showed a small pneumothorax at the left apex and an increase in the quantity of the air above the fluid level noted originally (Fig. 5).

Pain and occasional vomiting, present during the first 48 hours, subsided gradually. A low grade fever persisted. The patient remained ambulatory and was soon eating a general diet.

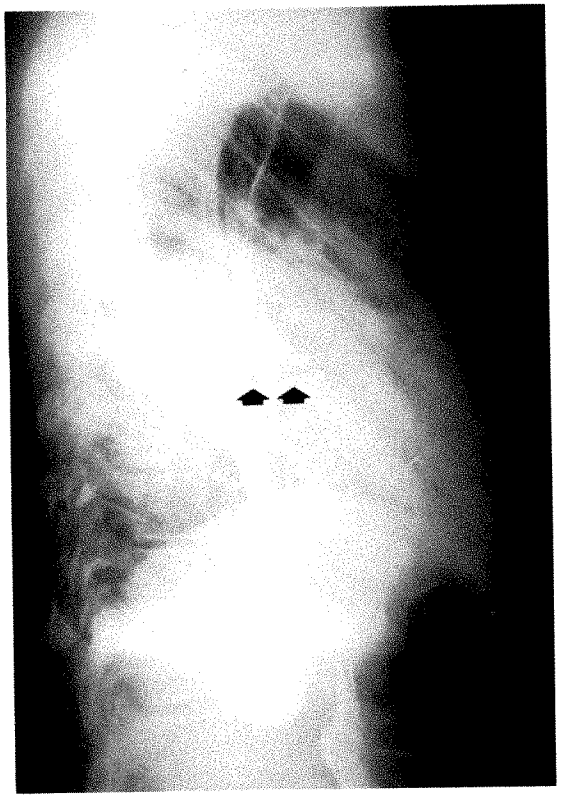


Fig. 3. Upright lateral roentgenogram of the chest showing the relationship of the fluid level to the esophagus (arrows) and the stomach.

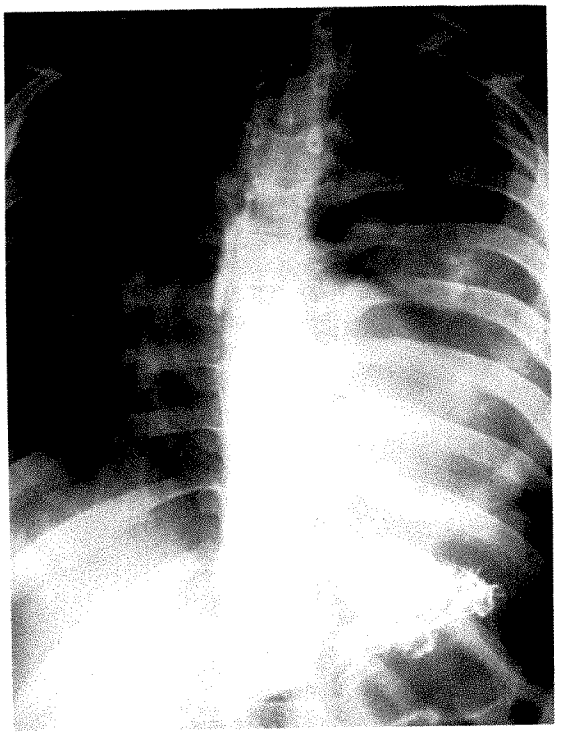


Fig. 4. Supine roentgenogram of the chest and upper abdomen with the patient in the Trendelenburg position showing the barium in the fundus and the "body" of the stomach.

The fourth day after admission, the hemoglobin dropped from 16.5 gm. to 14.1 gm. Eight days after admission the hemoglobin was 13.0 gm. A second attempt was made to demonstrate a hernia with gastrografin. In the Trendelenburg position the contrast material was seen to spiral upward from the fundus of the stomach through a small hole in the diaphragm into a portion of the body of the stomach incarcerated within the thorax. The lesser curvature portion remained within the peritoneal cavity and provided continuity of the bowel lumen (Fig. 6). Later that day, while awaiting surgery, the patient's temperature suddenly spiked to 104° F. Thoracotomy revealed acute perforation of the body of the stomach into the mediastinum. Approximately 70 per cent of the body of the stomach was resected and a 2 cm. rent in the diaphragm was repaired. After a stormy postoperative course,

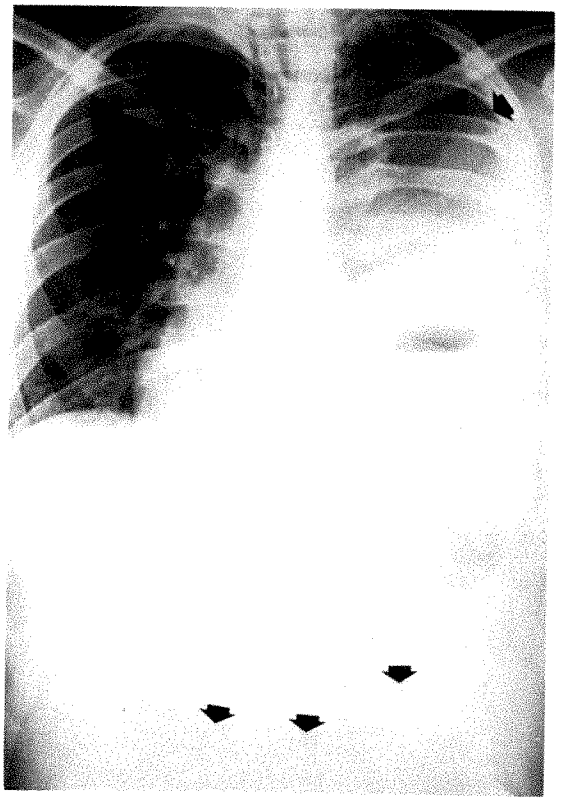


FIG. 5. Upright chest roentgenogram subsequent to thoracentesis shows a small quantity of air in the pleural space (upper arrow). Some residual barium is visible in the stomach and colon. Note the relatively normal position of the transverse colon (lower arrows).

the patient improved rapidly and was discharged 20 days after admission.

DISCUSSION

A Richter's hernia is one in which only a portion of the lumen is protruded.[7] The first recorded description of the condition was probably made in 1606.[11] Richter's description referred to a strangulation of small bowel in the femoral canal.[12] Theoretically any portion of the bowel capable of herniation through an orifice could produce a Richter's hernia; however, in overwhelming majority of reports, reference is made to hernias of the small bowel in the inguinofemoral region.[5,13] Cope[6] describes a case of femoral canal herniation of the cecum. Carter *et al.*[4] in illustrating the appearance of various gastric herniations did not include a Richter's hernia. This is, to our knowledge, the first such reported case. A review of larger surveys[1,8–10] fails to reveal a similar case.

In a recent article Blatt *et al.*[3] emphasize the frequency with which obstruction of a hiatal hernia is associated with a reversal of the pre-obstructive relationship between the proximal and distal stomach and the diaphragm. Bernatz *et al.*,[2] in a review of 112 cases of ruptured diaphragm, stress that a high index of suspicion is the most important factor in the diagnosis.

The presently reported case illustrates a pitfall in the "negative" gastrointestinal evaluation of a patient suspected of having a diaphragmatic hernia. It was felt that the small caliber of the defect in the dia-

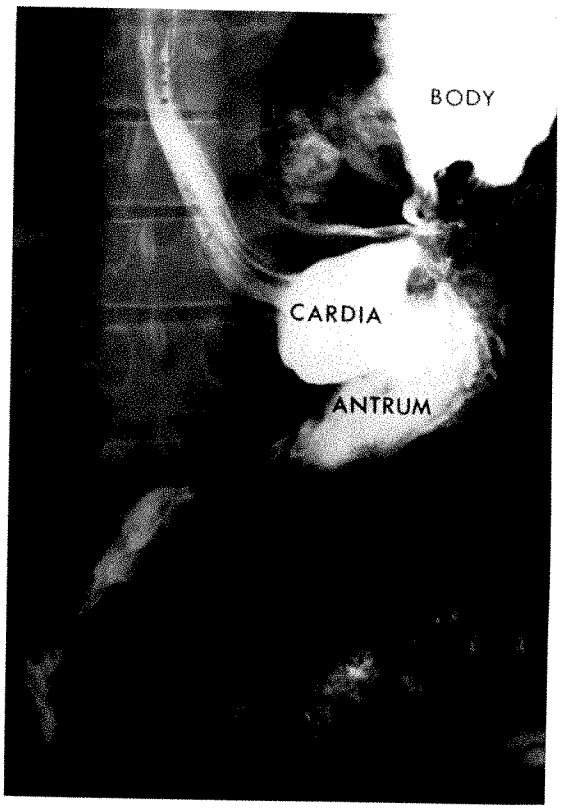


FIG. 6. Supine Trendelenburg roentgenogram of lower chest. Gastrografin can be seen spiraling upward into the twisted body of the stomach through a small rent in the diaphragm (arrow). The lesser curvature portion remains within the peritoneal cavity and provides the continuity of gastric lumen.

phragm coupled with gastric edema precluded earlier demonstration of the herniated portion. In retrospect, a diagnostic pneumoperitoneum might have been of value.

SUMMARY

A case of a strangulated Richter's hernia of the body of the stomach through a traumatic rent in the diaphragm is presented.

Appropriate roentgenographic studies generally verify or exclude the presence of an abdominal viscus within the thoracic cavity. In the case presented, the initial studies, while apparently excluding the diagnosis, led to a delay in treatment with nearly fatal complications.

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ISOLATED HETEROTAXY OF THE STOMACH*

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ISOLATED heterotaxy of the stomach is an exceedingly rare anomaly, there being only 10 prior examples reported in the world literature. Almy *et al.*, who contributed 1 case in 1953, reviewed the literature and found 6 earlier reports.[1] Since that time, there have been 3 additional cases described.[3,4,6]

In this abnormality the stomach lies behind the liver and is directed from right to left. As a consequence, the esophagogastric junction is on the right and posterior. The duodenal bulb also is directed from right to left and the ligament of Treitz is posterior and on the right. The other abdominal viscera are in their normal locations and the chest is also normal.

REPORT OF A CASE

W.S., a 40 year old Negro male, was admitted to Detroit Receiving Hospital with signs and symptoms suggesting an acute abdomen. He had been a consistent abuser of the grape over a period of years and any previous gastrointestinal symptoms had been related specifically to alcoholic bouts. Physical examination and laboratory studies were not remarkable. Roentgenographic examination of the chest showed no abnormalities with the heart being in its usual position. A gas shadow beneath the right diaphragm was noted (Fig. 1) and further roentgenographic examinations were made. An oral cholecystogram (Fig. 2) demonstrated that the liver and the gallbladder lay on the right in their normal positions, and that the spleen lay in its usual location on the left. Barium studies demonstrated an abnormal course of the distal esophagus, with the esophagogastric junction being located to the right of its usual position (Fig. 3). The stomach lay behind the liver (Fig. 4) and in a reversed position (Fig. 5). The duodenal bulb was directed from right to left. The ligament of Treitz was abnormally low and on the right side but distal to this the small bowel had its normal course. Follow-up roentgenograms demonstrated the colon to be normal with the cecum in normal position (Fig. 6). The gas shadow beneath the right diaphragm represented the stomach bubble.

A clinical diagnosis of alcoholic gastritis was made, and the patient was treated conservatively with complete remission of all symptoms within 2 days, after which he was discharged from the hospital.

DISCUSSION

Congenital right sided stomach occurs in many of the forms of abdominal heterotaxy ("situs inversus abdominalis") and may be associated with abnormalities of the heart, particularly in cases of isolated levocardia.[7,8]

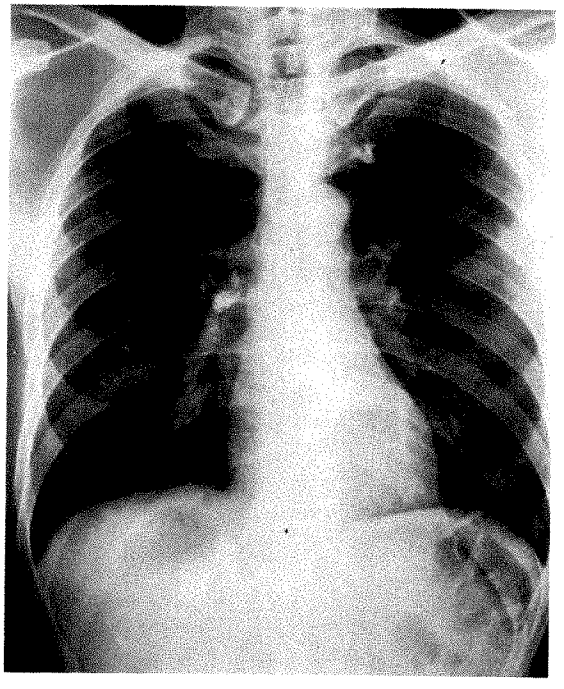
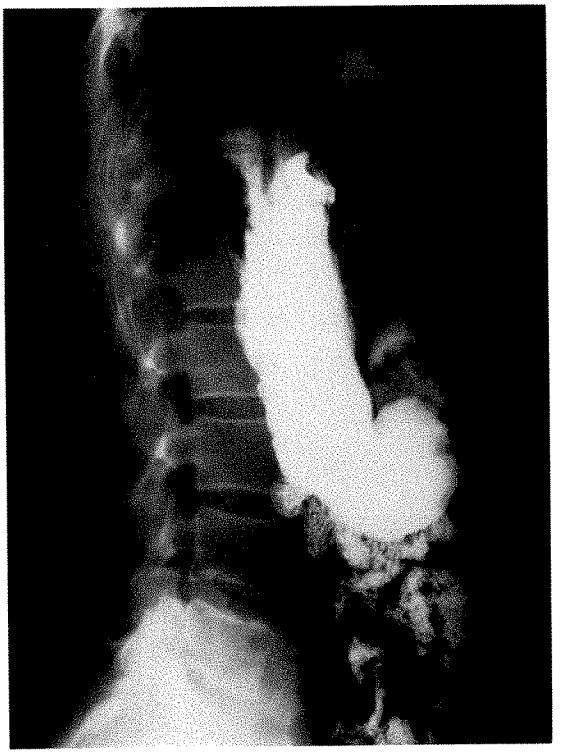


FIG. 1. Chest showing normal thoracic relationships and stomach bubble beneath the right diaphragm.

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Fig. 4. Lateral roentgenogram shows stomach to be behind the liver.



The stomach also may be displaced to the right by an abdominal mass or by adhesions.

There have been a number of cases reported of right sided stomach associated

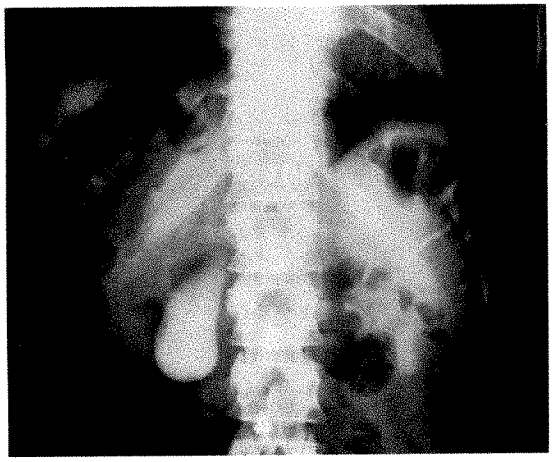


Fig. 2. Oral cholecystogram showing the liver and gallbladder on the right side, the spleen on the left side. Note the gas-filled stomach on the right side.

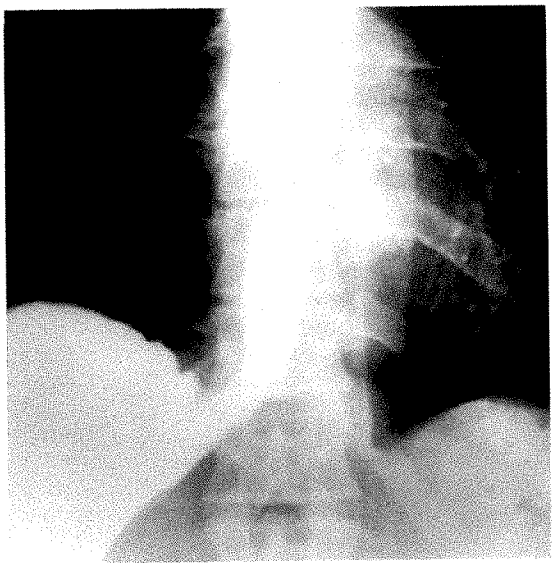


Fig. 3. The esophagogastric junction deviates to the right.

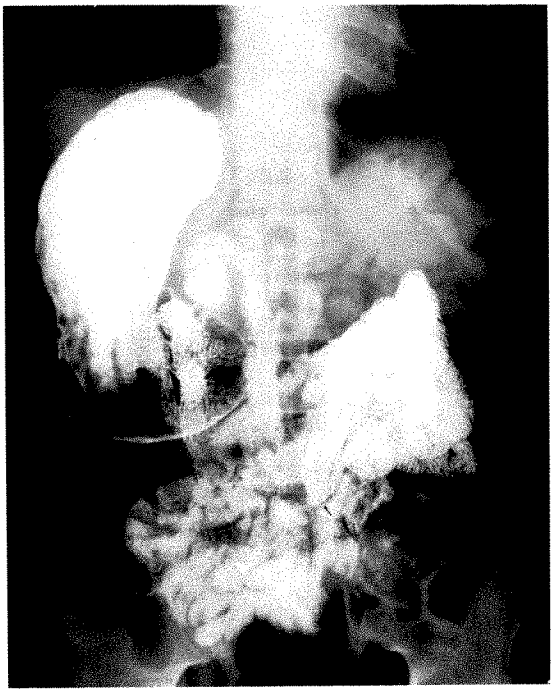


Fig. 5. Barium study shows right sided stomach with the duodenal bulb directed from the right to the left. The ligament of Treitz is also low and on the right side.

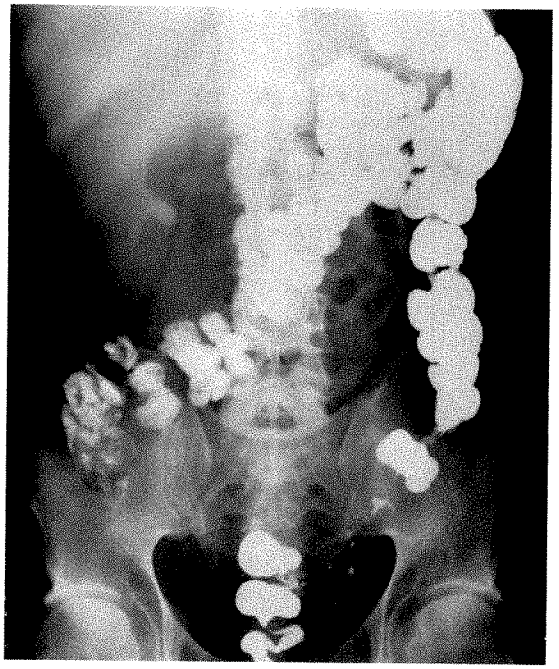


Fig. 6. The colon is normal except for slight depression of the hepatic flexure.

with eventration of the right diaphragm. In these cases, the stomach appears to be above rather than behind the liver and a different mechanism of development may well be involved.[3,5]

The differentiation of isolated heterotaxy of the stomach from the other anomalies is easily made, the key being the posterior location of the stomach, as well as the absence of other associated abnormalities.

The embryology of this abnormality is of considerable interest although the exact mechanism is not known. The stomach derives from the primitive foregut. Beginning as a simple midline tube in the thorax, the stomach rotates on its vertical and horizontal axes and descends into the abdominal cavity to its normal position. This usually has occurred by the end of the second fetal month. Reversal of normal rotation could account for dextroposition within the abdominal cavity.[2]

This portion of the intestinal tract does not become extruded into the umbilical cord, and therefore does not enter into the possible abnormalities of rotation involving the more distal portions of the bowel.

SUMMARY

An example of an unusual congenital anomaly, isolated heterotaxy of the stomach, is presented. The key feature of the abnormality consists of a right-sided stomach which is located behind the liver with the other thoracic and abdominal viscera being in their usual positions. This entity has no known clinical significance other than the fact that it exists.

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EOSINOPHILIC GASTROENTERITIS*

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EOSINOPHILIC infiltration of the stomach and small bowel occurs in two forms, which should be distinguished clinically, roentgenologically and pathologically.

The first is the so called "eosinophilic granuloma" of the stomach, a localized submucosal inflammatory polypoid lesion, characterized pathologically by discrete collections of fibroblastic, reticular and fibrillar connective tissue, and infiltration by chronic inflammatory cells, with eosinophils usually predominant.[22] Although usually located in the gastric antrum, occasional cases have been described in the proximal stomach, small and large bowel. The lesion is completely unrelated to the eosinophilic granuloma of bone, the basic cell type of which is the histiocyte, rather than the fibroblast. To the best of our knowledge, no case has been reported in which the two lesions coexisted. No specific clinical or laboratory findings have been described in this condition. The roentgen findings are equally nonspecific, and do not permit distinction from other polypoid or intramural lesions of the stomach. This entity has been adequately described[16,19,22] and is not the subject of the present paper.

The second, or diffuse type, has been previously reported under a variety of names, including the "gastric lesion of Loeffler's syndrome," "gastric granuloma with eosinophilic infiltration," "eosinophilic gastroduodenitis," and, unfortunately, "eosinophilic granuloma of the stomach." This confusion in terminology has resulted in a lack of adequate distinction between these two separate and distinct forms of gastrointestinal inflammatory disease. We prefer the term "eosinophilic gastroenteritis" for the diffuse type, which has sufficiently characteristic clinical, laboratory and roentgen features to permit diagnosis without resort to surgery.

We have encountered 2 cases of eosinophilic gastroenteritis at The New York Hospital. One of these has been previously reported.[23] Both patients, young males, presented with narrowed lesions of the gastric antrum and persistent blood eosinophilia. Biopsy revealed extensive eosinophilic infiltration of the stomach in one patient, and of the stomach, duodenum and jejunum in the other. A review of the literature revealed 22 similar previously reported cases (Table 1). It is the purpose of this paper to describe this entity, report an additional case with the unusual feature of protein losing enteropathy, and bring to the attention of radiologists the feasibility of clinical and roentgenologic diagnosis.

REPORT OF CASES

CASE 1. P. B., a 16 year old white male (N.Y.H. #940022), was transferred to The New York Hospital for evaluation of anemia, guaiac positive stools, hypoproteinemia and an abnormal upper gastrointestinal series. He complained of weakness, shortness of breath, and occasional regurgitation of clear fluid. He was otherwise asymptomatic. There was no history of allergies. Past history, family history and review of systems were noncontributory to the present illness. Physical examination revealed a well developed, well nourished white male who appeared pale, but otherwise well. There were no other abnormal physical findings. Significant laboratory findings were as follows: hemoglobin 6.1 gm. per cent, hematocrit 27.5, white blood cell count 14,200 with 14 per cent eosinophils; guaiac positive stools; total protein 4.7 gm. per cent with albumin/globulin 3.3/1.4 gm. per cent; serum iron 20 μg. per cent (5 per cent saturation). I^{131} labelled albumin turnover studies indicated protein loss into the gastrointestinal tract in excess of that which could be attributed to bleeding. Gastric chymotrypsin

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TABLE I

REPORTED CASES OF EOSINOPHILIC GASTROENTERITIS

References	Age	Sex	Symptoms	Roentgen Findings	White Blood Cell Count	Eosinophils (per cent)	Allergic History	Pathology
Kaijser[10] 1937	53	M	Pain, vomiting			26 (9 days postop.)	Onion intolerance	Eosinophilic infiltration in mucosa and submucosa
Herrera and de la Guardia[8] 1948	55	M	Pain, vomiting	Pyloric obstruction		Postop.		Muscular hypertrophy; eosinophilic infiltration in muscularis and submucosa
Barrie and Anderson[2] 1948	27	F	Pain, vomiting	Prepyloric filling defect; 6 hour retention	17,000	18		Muscular hypertrophy; eosinophilic infiltration in muscularis; para-arterial giant cells
Schneider and Dailey[18] 1948	64	M	Pain, hematemesis, melena	Antral narrowing simulating carcinoma; gastric retention	9,400	1	No	Eosinophilic infiltration most marked in submucosa
Moloney[14] 1949	57	F	Pain, vomiting		8,000	12		Muscular hypertrophy; eosinophilic infiltration in muscularis
Spencer *et al.*[20] 1950	40	M	Pain, vomiting, diarrhea	Pyloric obstruction	22,000	63	Urticaria	Muscular hypertrophy; eosinophilic infiltration in muscularis and submucosa
Doniach and McKeown[6] 1951	39	M	Pain, hematemesis	Rugal hypertrophy, particularly in antrum	5–10,000	5–10	No	Eosinophilic infiltration most marked in submucosa; muscle necrosis in muscularis
Barnett and Kaxmann[1] 1952	58	M	Epigastric distress, nausea	Antral deformity resembling carcinoma; gastric retention		Postop.	No	Muscular hypertrophy; eosinophilic infiltration in submucosa
Ruxic *et al.*[17] 1952	53	M	Pain, diarrhea	Huge lesser curvature filling defect	11,000	50	Asthma	Eosinophilic infiltration most marked in serosa, primarily perivascular
Orr *et al.*[16] 1954	38	F	Pain, vomiting	Normal		Postop.	Contact dermatitis	Muscular hypertrophy; eosinophilic infiltration in muscularis
	42	F	Pain, vomiting			Postop.	Many food allergies	Muscular hypertrophy; eosinophilic infiltration in muscularis
Judd *et al.*[9] 1955	34	M	Hematemesis	Smooth pyloric narrowing; gastric retention	24,600	54	Asthma	Muscular hypertrophy; eosinophilic infiltration in mucosa and submucosa
McCune *et al.*[12] 1955	28	F	Pain, vomiting, diarrhea	Pyloric obstruction	13,500	59		Eosinophilic infiltration in submucosa and muscularis
	54	M	Epigastric discomfort	Cone-like deformity of pylorus; gastric retention		34 Postop.		Muscular hypertrophy; eosinophilic infiltration in muscularis
	30	M	Vomiting	Prepyloric narrowing with ulcer-like crater	10,100	2		Eosinophilic infiltration and thickening of submucosa; no ulcer seen
Swarts and Young[21] 1955	34	M	Pain, vomiting	Pyloric obstruction	11,700	38	No	Muscular hypertrophy; eosinophilic infiltration in muscularis, submucosa and muscularis mucosae
	47	F	Pain, vomiting	Narrowed antrum	7,300	24	No	No gastric biopsy
Lynch *et al.*[13] 1956	35	M	Pain, vomiting	Pyloric obstruction	10,000	11	No	Muscular hypertrophy; eosinophilic infiltration in muscularis and submucosa
Ferrier and Davis[7] 1957	23	M	Pain, diarrhea	Dilated duodenum; clumping of barium in small bowel	13,000	21	No	No gastric biopsy; eosinophilic infiltration of small bowel and mesenteric lymph node
Cantor[4] 1959	40	F	Pain, vomiting	Narrowed antrum; dilated duodenum	5,000	10	Food intolerances	Thickened antrum and pylorus; eosinophilic infiltration in gastric wall
Kapp and Pagtalunan[11] 1961	64	F	Vomiting		11–22,000	62	Many, including penicillin	Eosinophilic infiltration between submucosa and muscularis
Weeks and Glenn[23] 1961	26	M	Vomiting	Pyloric narrowing and obstruction (Fig. 3)	14,500	23	Asthma, food intolerances, urticaria	Eosinophilic infiltration in mucosa and submucosa
Blackwell and Gild[3] 1962	55	F	Pain, vomiting	Narrowed pylorus and antrum; gastric ulcer	6,300	29	Numerous food intolerances	Muscular hypertrophy; eosinophilic infiltration in muscularis
Edelman and March 1964	16	M	Weakness, melena	Antral narrowing with "cobblestone" appearance (Fig. 1 and 2)	14,200	14	No	Pseudopolypoid mucosa; eosinophilic infiltration in mucosa, submucosa and muscularis

studies were Class 1. Gastroscopy showed a nodular appearance in the gastric antrum. Upper gastrointestinal and small bowel series revealed persistent narrowing of the distal antrum, with multiple polypoid filling defects having a "cobblestone" appearance (Fig. 1 and 2). No abnormalities were noted in the duodenum and small bowel. Gastric emptying was normal.

Following blood replacement, an exploratory laparotomy was performed. The pyloric portion of the stomach and the jejunum were thickened. Enlarged lymph nodes were present along both curvatures of the stomach and in the small bowel mesentery. A subtotal gastrectomy and gastroenterostomy were performed. The subsequent postoperative course was uneventful.

The mucosa in the resected antrum showed a pseudopolypoid appearance. The lamina propria and muscularis mucosae were completely infiltrated by eosinophils and plasma cells. Occasional eosinophils were present in the submucosa and muscularis. The mucosal changes extended into the duodenum. Similar changes were noted in a jejunal biopsy specimen. A resected lymph node showed hyperplasia of the reticuloendothelial cells and marked infiltration by eosinophils and plasma cells. No granulomata were seen. The pathologist's diagnosis was eosinophilic gastroduodenitis and jejunitis.

CASE II.* A.T., a 26 year old white male (N.Y.H. #362344), was admitted to The New York Hospital because of intermittent vomiting of food eaten many hours previously and inability to gain weight. Egg and wheat containing foods aggravated his symptoms. He had a past history of bronchial asthma, hay fever, and urticaria. There was a family history of allergy. Physical examination revealed an asthenic young male, with some tenderness to deep palpation in the epigastrium. No other abnormal physical findings were present. Significant laboratory findings were as follows: white blood cell count 14,500 with 23 per cent eosinophils; gastric analysis showed no free acid following histamine stimulation. Gastric chymotrypsin studies were Class 1. Upper gastrointestinal series showed persistent annular narrowing of the antrum with rigidity. The mucosa in the narrowed segment had a "cobblestone" appearance (Fig. 3). Gastroscopy revealed a reddened and granular antral mucosa.

* This case has been previously reported.[23]



FIG. 1. Case 1. Upper gastrointestinal study showing antral narrowing and multiple polypoid filling defects.

Dr. Frank Glenn's preoperative diagnosis was eosinophilic gastritis.

An exploratory laparotomy revealed thickening of the pylorus and slight enlargement of the mesogastric lymph nodes. Biopsy and frozen section revealed dense infiltration of the stomach with eosinophils. An anterior gastrojejunostomy was performed.

Histologically, there were many eosinophils in the lamina propria and muscularis mucosae with some involvement of the submucosa. There was edema of the gastric wall. The pathologist's diagnosis was eosinophilic gastroduodenitis.

Postoperatively, the patient had relief from vomiting and gained weight. A year and half later, he was re-admitted for massive gastrointestinal bleeding following generous alcoholic intake. A gastrointestinal series at this time showed a normally functioning gastroenterostomy and a narrowed pylorus. Whether the bleeding was related to persistent antral disease was not determined. He has since been well and symptom free.

DISCUSSION

Pathologically, eosinophilic gastroenteritis is characterized by thickening of the bowel wall and extensive infiltration by inflammatory cells, predominantly eosinophils. Most cases reveal maximal changes in the muscularis, with muscle hypertrophy and marked cellular infiltration between muscle bundles. Eosinophils predominate,



Fig. 2. Case I. Pressure spot roentgenogram of antrum showing "cobblestone" appearance to better advantage.

but mononuclear cells and other granulocytes are present in varying proportions. A tendency to perivascular location of the cellular infiltrate has been noted; occasionally, there is evidence of vasculitis. Others describe the infiltration predominantly in the mucosa, submucosa or serosa. It has been suggested that the layer of bowel wall chiefly involved may determine the presenting signs and symptoms; *i.e.*, muscle infiltration and hypertrophy may result in obstructive phenomena, while mucosal involvement may result in bleeding. A case of eosinophilic peritonitis reported by Swarts and Young[21] may reflect another manifestation of this same disease with predominantly serosal involvement. Our Case 1, the first reported case associated with a protein losing enteropathy, revealed maximal changes in the mucosa, lending support to this hypothesis.

An analysis of all cases reported to date, including our own (Table 1), reveals a nearly equal sex distribution, with an age range from 16 to 64 years (average age 42 years). Typically, these patients complain of epigastric pain and vomiting; occasionally, diarrhea and evidences of gastrointestinal bleeding are prominent features. Almost half of the patients give a history of allergies. *The most striking features, however, are an almost universal blood eosinophilia, and roentgenographic evidence of pyloric and antral narrowing and obstruction.* All reported cases with positive roentgenographic findings showed antral disease. Small bowel involvement frequently accompanies the more clinically and roentgenographically evident gastric lesion.

Although distinction must be made from other conditions which may narrow and obstruct the gastric outlet, the combination of antral narrowing and blood eosinophilia (as high as 60 per cent), particularly in a young patient with an allergy history, should alert the radiologist to the possibility of this uncommon disease.

The diagnosis is of more than academic interest, as optimal treatment for eosinophilic gastroenteritis is conservative. The disease is apparently self limited, and may be characterized by remissions and exacerbations. A number of reported cases gave a history of recurrent symptoms similar to those which later led to further investigation and diagnosis. In the case reported by Ruzic *et al.*[17] an upper gastrointestinal series and eosinophil count returned to normal

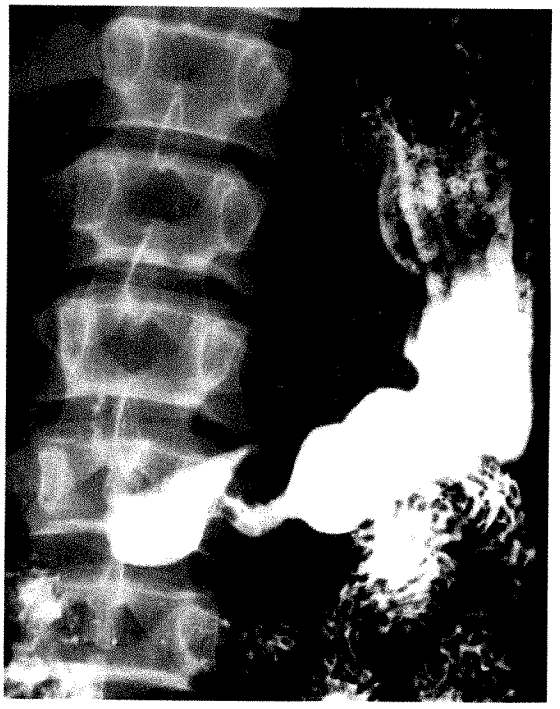


Fig. 3. Case II. Upper gastrointestinal study showing marked narrowing of the gastric antrum with multiple small filling defects.

in 3 months, coincident with resolution of symptoms. Other authors have emphasized the tendency to spontaneous remission.[3,7,20,21] Prompt clinical improvement has been reported following ACTH therapy.[7,15] If surgery is performed, either because of inability to reasonably establish the diagnosis or to relieve pyloric obstruction or bleeding, the most conservative procedure (*i.e.*, biopsy, simple gastroenterostomy) should be done. Gastric resection would not appear indicated, except to control hemorrhage. In cases in which a biopsy was taken and the lesion left *in situ*, a satisfactory result was obtained.[14,17,23]

The etiology of eosinophilic gastroenteritis is unknown. Whether eosinophilic infiltration of the bowel wall reflects a nonspecific host response to a variety of stimuli in allergic individuals, or is, in fact, a single disease entity with varied manifestations is speculative. The possibility that this may represent a localized gastrointestinal manifestation of allergy has been suggested by most authors, but no consistent relationship between exposure to a specific allergen and symptomatic exacerbation has been established. Parasitic etiology has never been proven. The typically benign course militates against a relationship to a collagen vascular disease of the polyarteritis nodosa type. In spite of our present inability to document a common etiology and pathology in the cases reported to date, there is little doubt that eosinophilic gastroenteritis forms a recognizable clinical and roentgenographic entity which often can be diagnosed without resort to surgery.

SUMMARY

Eosinophilic gastroenteritis is described and an additional case with the unusual feature of protein losing enteropathy is reported. Attention is called to the feasibility of clinical and roentgenographic diagnosis.

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VOL. 91, NO. 4

CALCIFICATION WITHIN A GASTRIC CARCINOMA*

A CASE REPORT

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THAT there is a great variety of abdominal calcifications of varying etiology is well known. Among these, calcifications within gastrointestinal neoplasms occur infrequently. Benign gastric neoplasms, such as leiomyomas, sometimes show calcific deposits. Hemangiomas of the gastrointestinal tract are also known to calcify. Calcifications within malignant gastrointestinal tumors are, however, exceedingly rare. Several cases of calcifying colon carcinomas have been reported. Only 5 proved cases of calcifications within gastric carcinomas have been described in the English medical literature; the foreign literature is likewise scant on this subject.

The author's purpose is to present a case of gastric carcinoma in which calcium deposits were demonstrated on roentgenologic examination. This permitted a correct preoperative diagnosis. As far as we could determine, this is now the sixth such case reported in the English medical literature. One of the other cases was also reported from this department by Batlan.[2]

REPORT OF A CASE

A 59 year old Negro male was admitted to Michael Reese Hospital on April 2, 1962 complaining of a 30 pound weight loss in 8 months, combined with marked loss of appetite. There was no hematemesis or melena. Physical examination revealed a firm, nontender mass in the epigastrium and questionable hepatomegaly. The cholecystogram showed a normally functioning gallbladder; no stones were visualized. The barium enema study was normal; however, there were rather well defined punctate densities noted in the stomach area (Fig. 1). The barium meal studies revealed marked deformity of the body of the stomach in association with a mass. The calcific densities were again observed within the markedly thickened and rigid gastric wall (Fig. 2, *A*, *B* and *C*). Because of the calcifications, mucinous adenocarcinoma of the stomach was diagnosed. Gastroscopy disclosed a lesion suspected of being malignant. Cell washings were positive for carcinoma. At surgery an extensive carcinoma of the stomach was found, involving the entire body as well as the fundus. There were metastases to the liver, omentum and peritoneum. Biopsy of one of the omental lymph nodes showed mucinous adenocarcinoma (Fig. 3). No gastric resection was performed.

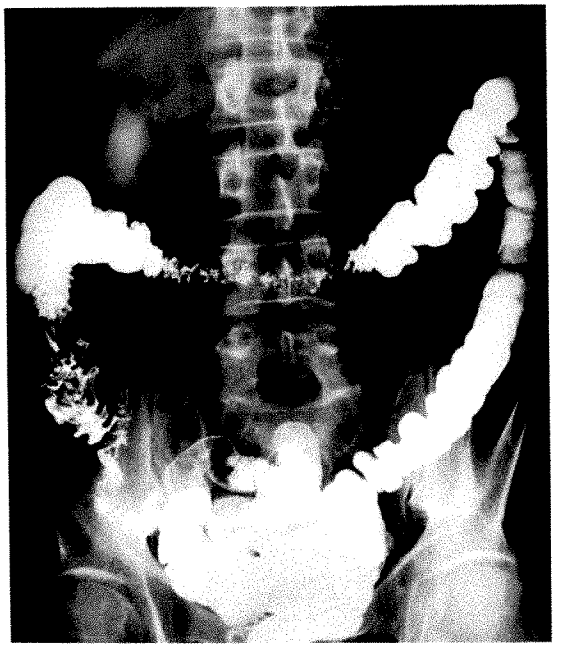


FIG. 1. Barium enema study showing calcifications in the area of the stomach.

DISCUSSION

Pathologists are well aware of calcifications within stomach cancers and mention of these is made in *Virchow's Archives* as early as 1928. However, since there was no correlation with roentgenologic findings,

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we shall not concern ourselves with these reports.

The roentgenographic demonstration of calcium deposits within an adenocarcinoma of the stomach is exceedingly rare. Various theories as to the cause have been proposed of which the theory regarding the so-called dystrophic calcifications seems the most plausible.

These are calcifications in ischemic and

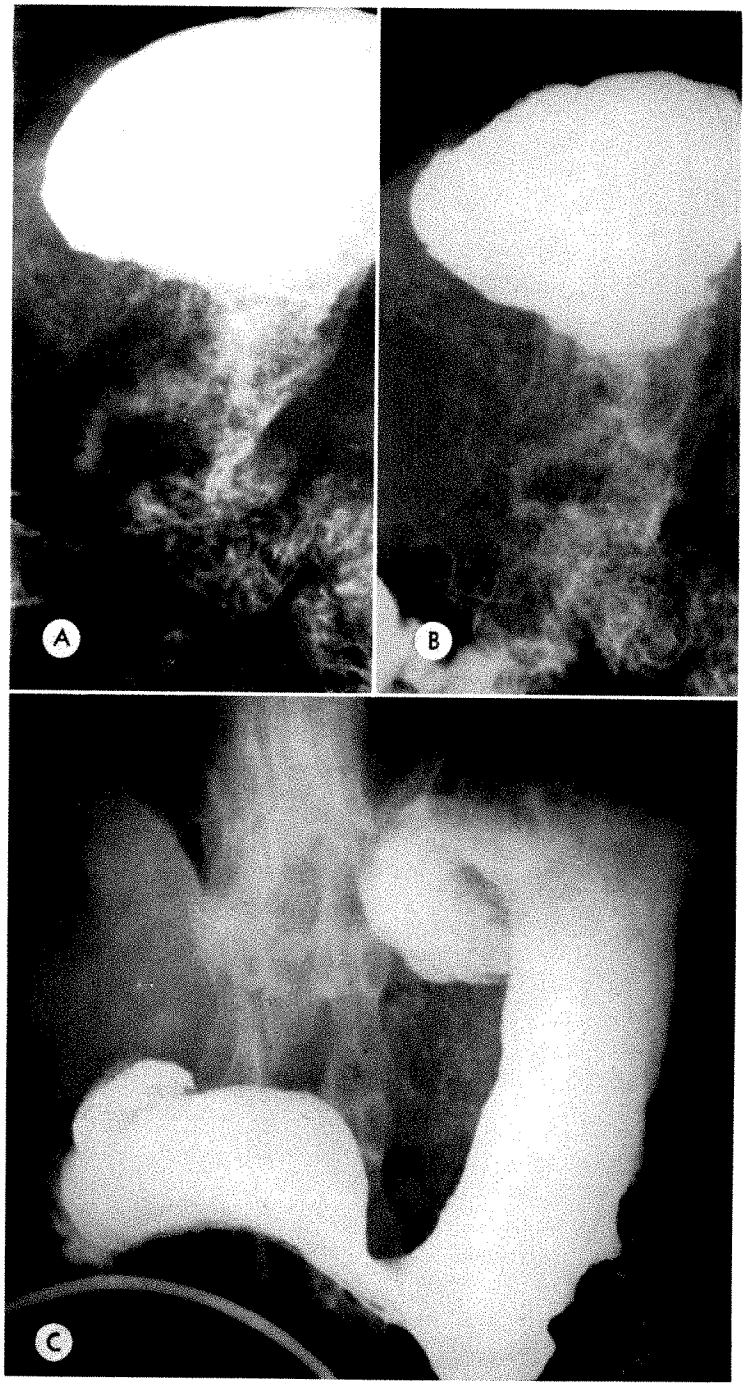


Fig. 2. (*A* and *B*) Supine spot roentgenograms of the stomach showing calcifications within the thickened gastric wall. (*C*) Calcifications well demonstrated within gastric wall.

TABLE I

ANALYSIS OF CASES OF MUCINOUS ADENOCARCINOMA OF THE STOMACH WITH ROENTGENOLOGICALLY DEMONSTRATED CALCIFICATIONS

Author	Year	Sex	Age	Clinical Findings	Roentgenologic Findings
Beliczay[3]	1937	F	35	Ascites; extensive infiltration; distant metastases	Coat-like gastric calcifications
Abel[1]	1939	F	30	Ascites; palpable mass; mucous producing carcinoma involving most of the stomach	Mottled calcifications throughout thickened stomach wall
Riedel[13]	1940	F	40	Palpable mass; mucinous adenocarcinoma involving entire stomach	Stippled calcium deposits in left upper abdomen
Tenconi[16]	1948	M	37	Anterior wall mucinous adenocarcinoma	Calcific densities along lesser curvature
	1948	M	42	Mucinous adenocarcinoma involving antrum	Antral neoplasm with mottled calcific densities
	1948	M	51	Ascites; antral mucinous adenocarcinoma	Lesser curvature mottled calcifications
Robertson and Osterhout[14]	1952	M	75	Moderately well differentiated mucinous adenocarcinoma involving distal half of stomach; regional lymph node metastasis	Poorly outlined mottled calcific shadow associated with mass
Batlan[2]	1954	M	47	Mucinous adenocarcinoma involving distal two-thirds of stomach	Infiltration of lower stomach, stippled calcium deposits along lesser curvature and posterior wall
Kendig[7]	1957	F	27	Mucoid carcinoma simplex with regional lymph node metastases	Subtotal resection with recurrence of tumor with calcifications
Matthews[11]	1958	F	59	Mucinous adenocarcinoma involving distal two-thirds of stomach	Punctate and flake-like calcifications
Petersen[12]	1958	M	77	Extensive infiltration of pars media and distal portion; mucinous adenocarcinoma	Numerous granular calcifications
Khilnani[8]	1960	M	63	Huge mucoid adenocarcinoma involving the entire stomach; regional and distal metastases	Innumerable small calcifications in left upper quadrant of abdomen, associated with mass
Present Author	1963	M	59	Mucinous adenocarcinoma involving fundus and body of stomach	Punctate calcifications projected upon upper part of stomach

necrotic areas, which are said to occur because of the relative alkalinity of the circulating extracellular fluid—brought about by diminished cellular respiration and diminished carbon dioxide production, a situation that may result from insufficient blood supply. The balance between calcium solubility and deposition as the salt de-

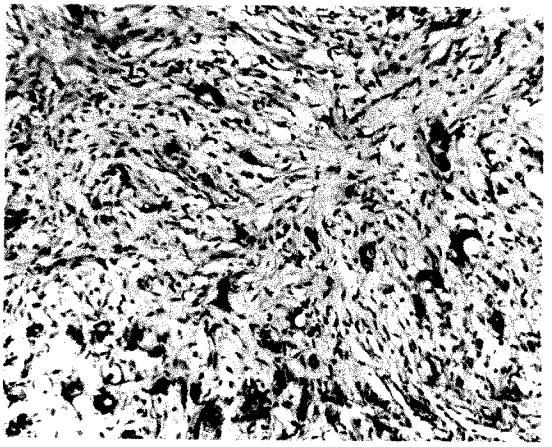


Fig. 3. Omental lymph node biopsy showing mucinous adenocarcinoma (×150, H & E).

pends largely on the pH of the medium, increasing as the pH increases.

Why only a very small percentage of gastric cancers calcify is not known. Of interest is the fact that almost all of the calcium-producing gastric cancers including the reported case were mucinous adenocarcinomas. The only other tumors of similar histology that occasionally calcify are the ovarian papillary mucoid and cystic mucoid adenocarcinomas, as well as mucoid colon carcinomas. The former may show the rather characteristic granular calcifications, so-called psammocarcinomas.

The appearance of the calcifications is sufficiently characteristic to permit diagnosis from plain roentgenograms with reasonable accuracy. In the differential diagnosis one would have to consider calcifications within the pancreas, spleen, left kidney and adrenal which are generally quite different in appearance. Retained gallbladder contrast medium might be confusing.

Table 1 is a summary of the clinical and roentgenologic findings in cases heretofore reported. It is of special note that in all but one case of calcified gastric cancer the tumor was mucinous adenocarcinoma.

Leiomyomas of the stomach commonly exhibit degenerative changes including fibrosis, hemorrhage, hyaline degeneration, and calcification. This histologic appearance is said to resemble uterine leiomyomas. It is estimated that only 4 per cent of all leiomyomas reported in the literature have shown calcifications roentgenologically. The calcific densities within the mass are usually circumscribed, stippled, or patchy and at times quite mobile. An intraluminal mass can invariably be demonstrated by a gastrointestinal series with localized deformities of the gastric wall. Extremely small tumors, 1–2 cm. in diameter, have been detected on roentgenograms by virtue of the calcification.

SUMMARY

A case of calcification within a gastric mucinous adenocarcinoma has been presented. Only 5 other proved cases have been described in the English medical literature. The calcium deposits consist of small stippled and flake-like concretions and are sufficiently characteristic in appearance to make possible the correct diagnosis from a plain roentgenogram of the abdomen.

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RADIOACTIVE I^{131} AND P^{32} AS AIDS IN THE DIAGNOSIS OF LESIONS OF THE STOMACH*

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WHILE there are undoubtedly physicians who subscribe to the doctrine of immediate surgical excision for all gastric lesions, the majority prefer to obtain as much diagnostic information as possible with a view to selection of those patients who may be safely treated by nonsurgical means. In order to do this with confidence, the various diagnostic measures employed should achieve a high degree of reliability.

Even among those who advocate the immediate removal of all demonstrated and suspected gastric lesions, the preoperative confirmation of benignancy or malignancy is most helpful in avoiding operative biopsy and in assisting the planning of a rational operation. Based on Dragstedt's[5] implication of antral stasis as the basic mechanism in the etiology of benign gastric ulcer, there is a small but increasing trend to treat apparently benign gastric ulcer by vagotomy and pyloroplasty, with or without wedge resection of the ulcer.[6] In fact, the high and difficult juxta-esophageal ulcer has long been treated on these principles.[8] While there is almost universal agreement that biopsy of the ulcer is mandatory, decision on the frozen section histology is not always easy. Any method, therefore, providing evidence in the direction of benignancy or malignancy is valuable.

Furthermore, increased longevity in the general population may be expected to lead to an increasing number of patients with concomitant emphysema, cardiovascular disease and gastric lesions. Many of these patients with poor pulmonary function and cardiac reserve tolerate general anesthesia poorly and operation is often hazardous. It is consequently of greatest importance to attempt to separate benign and malignant lesions in this group.

Unfortunately, it must be recognized that conventional roentgenologic studies, while virtually diagnostic in many cases, may occasionally be equivocal or misleading even in the best hands. Radiologists are familiar with the malignant ulcer masquerading as a typical peptic ulcer—and vice versa— the "indeterminate" ulcer, the problem of locally or generally enlarged folds, and with the difficulty in differentiating the causes of a narrowed antrum, to cite a few common examples. Skillful gastroscopy and exfoliative cytology may help to resolve some, but not all of these problems. Each method has its definite, well-recognized limitations, and there can be no question that any safe, reliable, and easily performed investigation which gives corroborative evidence will be helpful and may, in fact, under certain special clinical circumstances, become invaluable.

In the hope of improving the radiologic diagnosis of gastric lesions, we have investigated the clinical value of NaI^{131} and P^{32} gastric uptake in various benign and malignant lesions of the stomach. Contrary to a previous report,[4] we found no selective uptake of NaI^{131} in 12 gastric carcinomas studied preoperatively by means of scintiscans of the stomach and we concluded that this approach has no clinical merit. Figures 1, 2, 3 and 4 show the findings in an ulcerating carcinoma of the stomach and are representative of the results in all the cancers studied.

Selective uptake of P^{32}, however, in gastric cancer as well as in various other malignant neoplasms is an established

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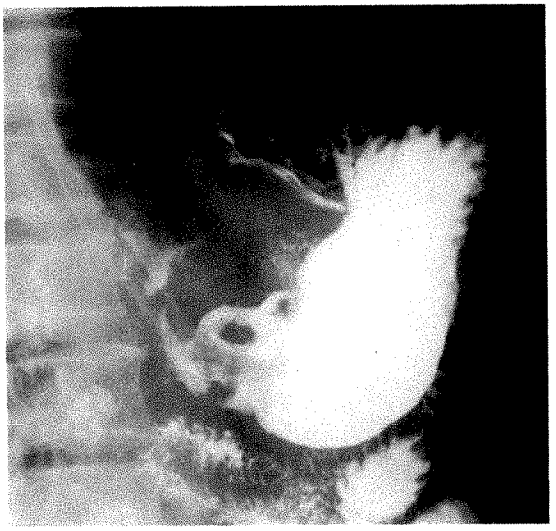


Fig. 1. Large saucer shaped ulcer of lesser curvature with nodular mucosal border suggesting carcinoma.

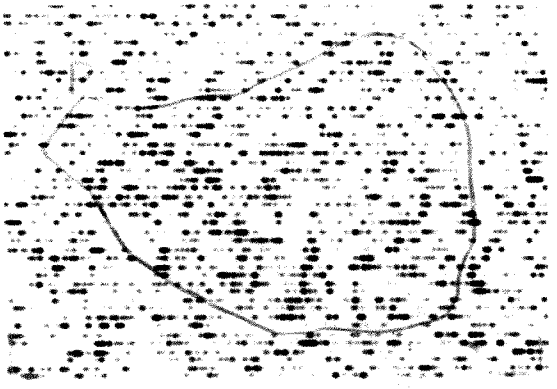


Fig. 3. Scintiscan of gross specimen approximately 3 hours after 500 μc NaI^{131}. No selective uptake. "P" represents pyloric end of specimen.

fact.[7,9,10,15] Nucleoprotein and lipid phosphorus metabolic rates are at least 45 per cent higher in human gastric cancer than in noncancerous mucosa,[12] and measurements of resected stomach cancers have shown P^{32} uptake increased in the ratio of 1.3:1 to 13.5:1 compared to normal mucosa.[14]

Nakayama[11] was the first to use this biochemical feature of neoplasms in the clinical diagnosis of carcinoma of the stomach. His approach consisted of comparing counts of normal mucosa with a lesion using small Geiger-Müller tubes placed adjacent to the suspected area under fluoroscopic guidance, 6 to 48 hours after 300–500 μc of P^{32} had been given intramuscularly. Ratios of 1.3:1 or higher were found in malignant lesions.

Several years later Ackerman *et al.*[1,2] devised a method called "*in vivo* radioautography" in which a rubber balloon coated with an elastic, latex-base photosensitive emulsion attached to a nasogastric tube was placed in the stomach 2 hours after

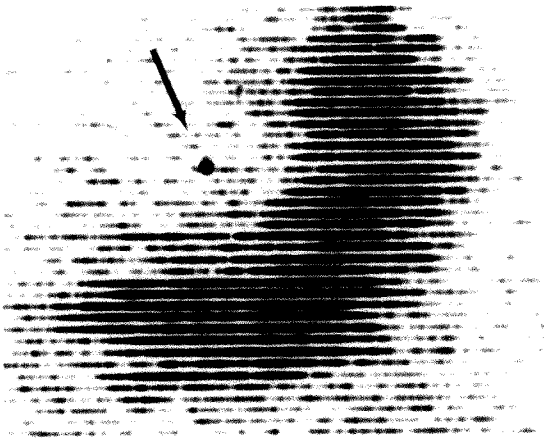


Fig. 2. Three hour scintiscan of stomach after 500 μc of NaI^{131}. There is no selective uptake in the lesion (heavy black dot at left center represents xiphoid process).

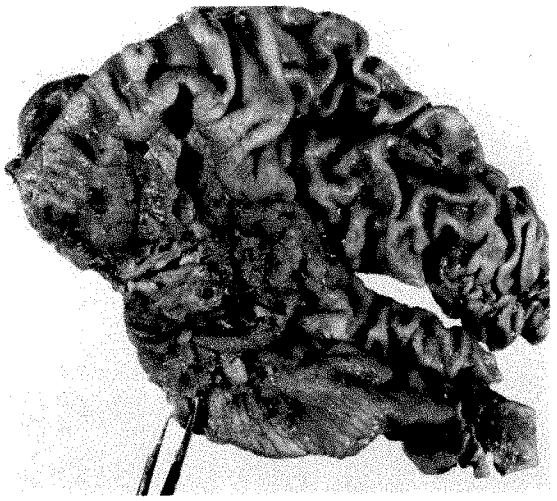


Fig. 4. Photograph of specimen opened along greater curvature. The pylorus is to the left. Histologic diagnosis: adenocarcinoma, ulcerating.

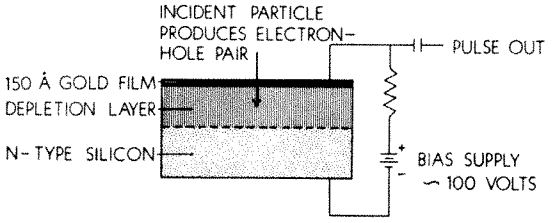


FIG. 5. Schematic drawing of surface barrier type of solid state detector. See text for principles of operation.

500 μc of intravenously administered P^{32}. With this technique phosphorus radioactivity in carcinoma could be detected by the density produced on the developed emulsion.

In our study we have used a solid state detector, placed in the stomach under fluoroscopic or gastroscopic control, as the measuring instrument to detect phosphorus radioactivity. We believe that this approach possesses the advantages of simplicity, high counting efficiency, and reliability; and that it can provide valuable information important to the clinical management of a patient with a gastric lesion.

THE SOLID STATE DETECTOR[3,13]

A solid state detector is analagous to an ionization chamber. Both are instruments for detecting radiation, but the function of the gas in an ionization chamber is performed in the solid state detector by semiconducting solid materials.

Figure 5 shows schematically the structure of one type of solid state detector, the surface barrier type. A wafer of specially prepared silicon has diffused into its surface a dissimilar substance to produce a junction between two semiconducting materials, the so-called "p-n" junction. A thin surface layer of gold provides a uniform electric field across the junction. For radiation detection, the detector is operated with a reverse voltage polarity, or bias, which establishes an electric field and a radiation-sensitive volume on either side of the junction. This is called the depletion layer. It is in this region that radiation interacts to form "ion-pairs," or, in the language of solid state physics, electron-hole pairs.

When a charged particle (*e.g.*, the beta particle of P^{32}) enters the energized detector, electron-hole pairs are formed by displacement of electrons from their sites in the depletion layer. The electrons drift toward the positive electrode, and the positively charged regions or "holes," which remain in the wake of the displaced electrons, migrate toward the negative electrode. Thus each charged particle entering the detector causes a flow of charge resulting in a pulse, or count, the magnitude of which is proportional to the energy of the interacting particle.

The detector is connected to a charge-sensitive pre-amplifier for maximum sensitivity. The output of this amplifier is handled by conventional amplifiers, scalers, and rate meters.

Several characteristics of solid state detectors make them extremely useful. Among these are: (1) small size—the sensitive area of the surface barrier type used in this study is 7 sq. mm.; (2) ruggedness; and (3) high sensitivity—free mobility of the charged carriers in the depletion layer and an extremely short distance between electrodes results in collection times as short as 10^{-9} seconds. Furthermore, 100 per cent efficiency in counting charged particles can be achieved if the depletion layer is thick enough.

CLINICAL MATERIAL AND METHOD

Fifty-three patients with roentgenologically demonstrated or suspected gastric lesions were examined. With the exception of 4 cases of peptic ulcer treated medically and followed to complete clinical and radiologic healing, a histologic tissue diagnosis was obtained in each patient.

A solid state detector—the end-sensitive, cylindrical, or rectangular type—was attached by subminiature coaxial cables to the rate meter. A spring loaded deflector and thin hollow polyethylene tubing were placed adjacent to the cable and the entire apparatus encased in RTV-type silicon

rubber covering, leaving an outlet for the polyethylene tubing so that air could be pumped into the stomach (Fig. 6).

Four hundred microcuries of P^{32} were given intravenously, 18 to 24 hours prior to the examination. The tube containing the detector was passed into the fasting stomach after anesthetizing the pharynx. Using image intensified fluoroscopy, the stomach was inflated with air, and the detector was placed first against the wall of normal mucosa and then against the lesion as determined by previous roentgen studies. In a number of cases the detector was positioned under direct vision during gastroscopy.

Since the average energy of the beta radiation from radioactive phosphorus is 0.695 mev. with a maximum tissue penetration of 0.8 cm. and length of travel in air of 10 cm., the detector was carefully positioned with the sensitive portion of the probe adjacent to the area being counted so as to avoid erroneous count rates due to the inverse square law. Usually, this could be accomplished by rotation of the patient in the erect, prone and supine positions and by the use of the deflector. Several determinations were made in the area in question to be certain the lesion itself was counted. The entire procedure, on the average, required no longer than 10 to 15 minutes.

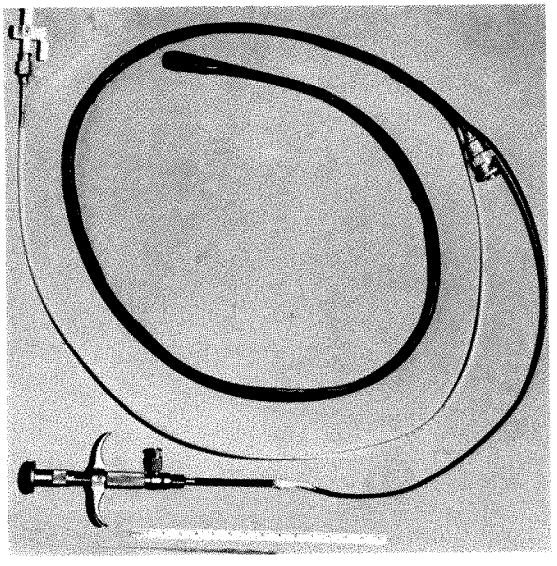


Fig. 6. Photograph of apparatus. The detector (end-sensitive type) is at the tip. The cables are attached to pre-amplifier and rate meter through connection at upper right. The deflector is manipulated with spring at bottom. The polyethylene tube connector is at upper left.

Table I

Results of P^{32} uptake in benign and malignant gastric lesions

Final Diagnosis	No. of Cases	Differential Ratio*	Per Cent Correct
Carcinoma†			
Ulcerating	5	1.5:1 to 3.0:1	
Polypoid	3	(median	93.8
Infiltrating	3	1.8:1)	
Mixed	5		
	16		
Benign‡			
Peptic Ulcer	26	1:1 to 1.25:1[z]	97.3
Pyloric Hypertrophy	2		
Leiomyoma	2		
Adenoma	1		
Miscellaneous	6		
	37		
Total	53		96.2

* Count rates of lesion relative to normal mucosa.
† Includes one false negative.
‡ Includes one false positive.
[z] Only 2 patients with peptic ulcer had ratios of 1.2:1 and 1.25:1, respectively.

In several patients later proven to have benign peptic ulcers, venous blood from the arm was withdrawn, counted, and then injected into the stomach to test the effect on the count rate of radioactive blood in the lumen. A second count rate was then determined and compared with the first.

Results

With a dose of 400 μc of P^{32}, the average count rate of normal gastric mucosa at 18 to 24 hours was 100/minute.

The results of the differential counts in 53 patients are summarized in (Table 1). In the benign lesions including peptic ulcer and miscellaneous conditions, the differen-



tial count ratios ranged from 1:1 to 1.25:1. Two cases of histologically proven benign peptic ulcers showed differential ratios of 1.2:1 and 1.25:1, respectively; in the remainder the ratio was 1:1.

In patients with cancer of the stomach, the ratios ranged from 1.5:1 to 3.0:1 with a median of 1.8:1.

One false positive and one false negative were encountered.

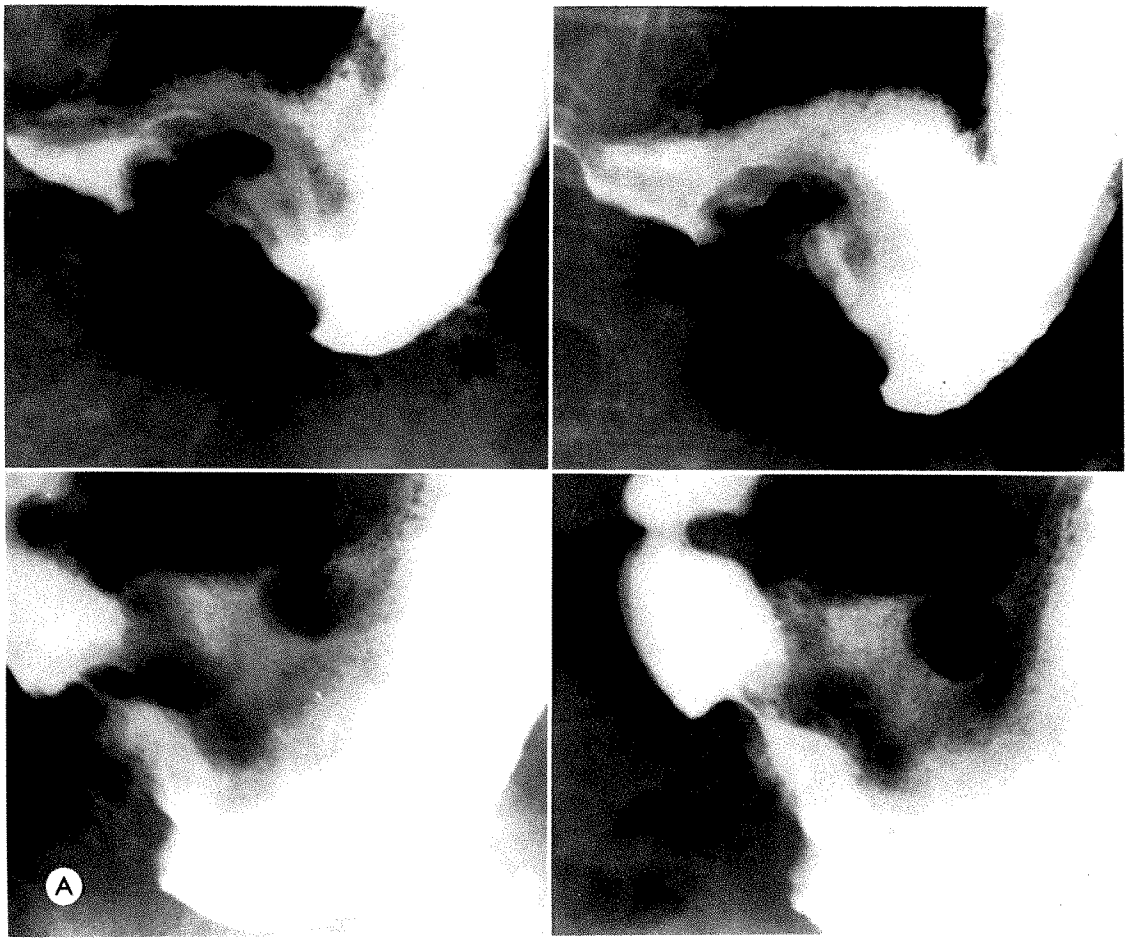
The patients with benign ulcer in whom 20 cc. of venous blood was injected into the stomach showed no alteration in the count rate.

ILLUSTRATIVE CASES

Case I. Fig. 7, *A–C*. An 80 year old white male with symptoms of easy satiety and immediate postprandial epigastric pain for 6 months was thought to have an ulcerating carcinoma, roentgenologically. Free acid was present in the stomach and exfoliative cytology revealed no malignant cells. P^{32} uptake ratio of 1.7:1 confirmed the impression of carcinoma. Pathologic diagnosis: polypoid adenocarcinoma, ulcerating.

Case II. Fig. 8, *A–C*. An 81 year old white female was examined because of massive gastrointestinal bleeding requiring two units of blood. A polypoid mass with central ulceration, typical of carcinoma, was present on the greater curvature of the antrum. In addition, a 2 cm. sessile polypoid lesion was demonstrated at the incisura angularis. Both lesions had differential uptake ratios of 1.6:1 and 1.8:1, respectively. Histologically, the greater curvature lesion was an ulcerating adenocarcinoma; the polypoid lesion was a noninfiltrating adenocarcinoma, Grade 1. This case illustrates the possibility of distinguishing a benign from a malignant small polypoid lesion.

Fig. 7. Case 1. (*A*) Large flat ulceration in distal antrum surrounded by polypoid filling defect suggesting ulceration within mass. Differential uptake 1.7:1 confirmed impression of carcinoma. (*B*) Photograph of resected specimen showing polypoid adenocarcinoma with central ulceration. (*C*) Autoradiograph of specimen. Heavy concentration of radioactivity in non-ulcerated portion of tumor. Note submucosal metastases represented by small black areas to right of main tumor.



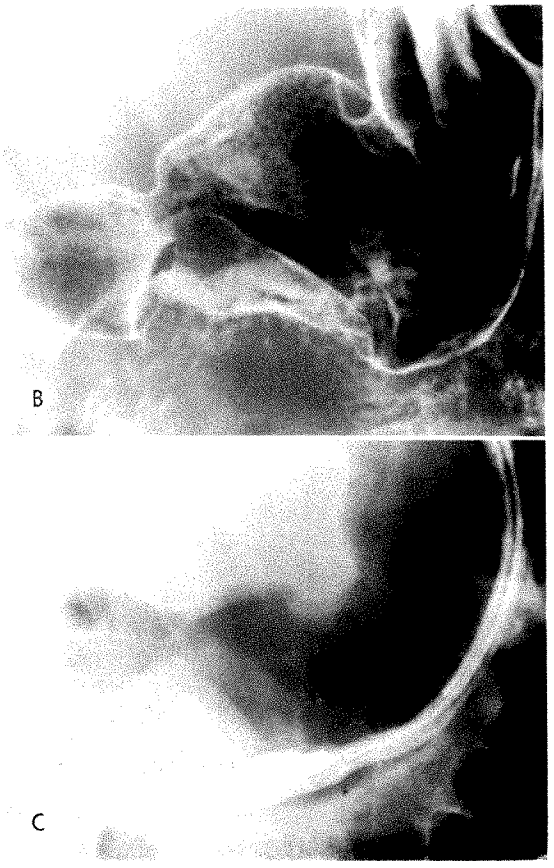


FIG. 8. Case II. (*A*) Spot roentgenograms of antrum. Ulcerating mass along greater curvature is typical of carcinoma. Sessile polypoid lesion at incisura angularis. (*B*) Spot roentgenogram of the antrum and (*C*) of the detector against the greater curvature lesion. The detector was also placed against the polypoid lesion. Ratios of 1.6:1 and 1.8:1, respectively, were obtained. Histology of ulcerating lesion: adenocarcinoma, ulcerating and infiltrating; that of polypoid lesion also adenocarcinoma, noninfiltrating.

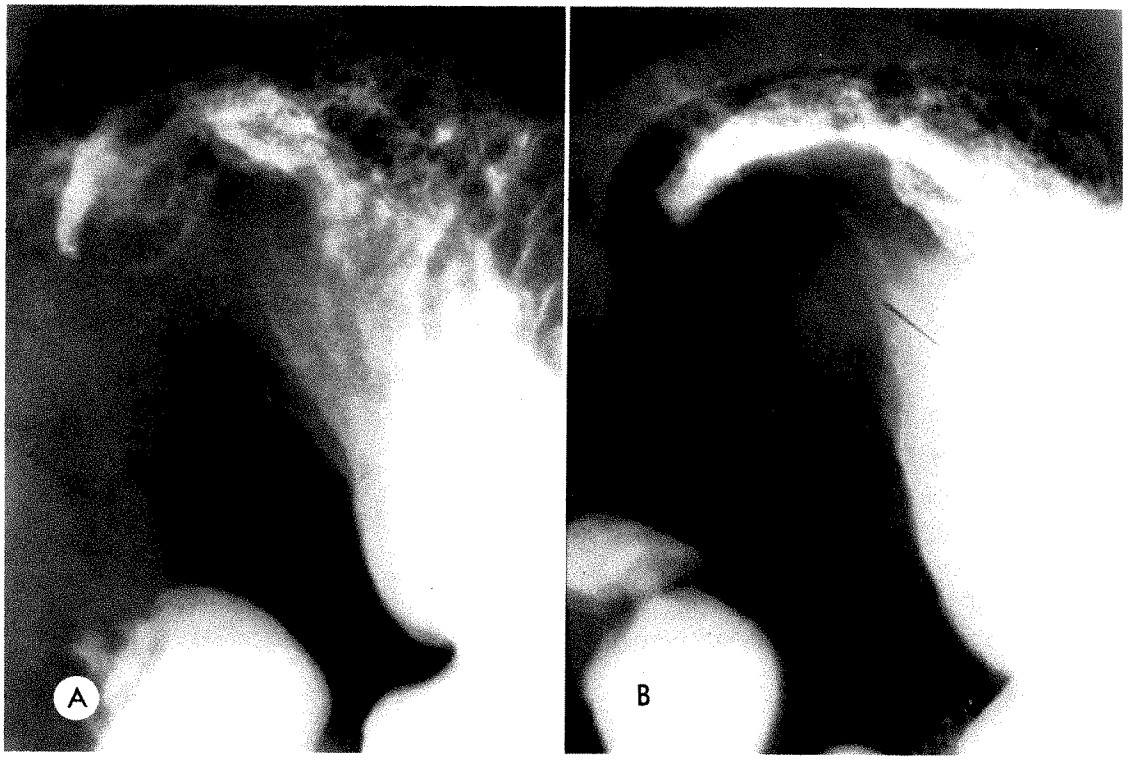


Fig. 9. Case III. (*A* and *B*) A large mass with multiple areas of ulceration is present on the posterior wall at the level of the cardioesophageal junction. With the fundus distended (*B*), the upper border is smooth. Clinical diagnosis: carcinoma of the stomach.

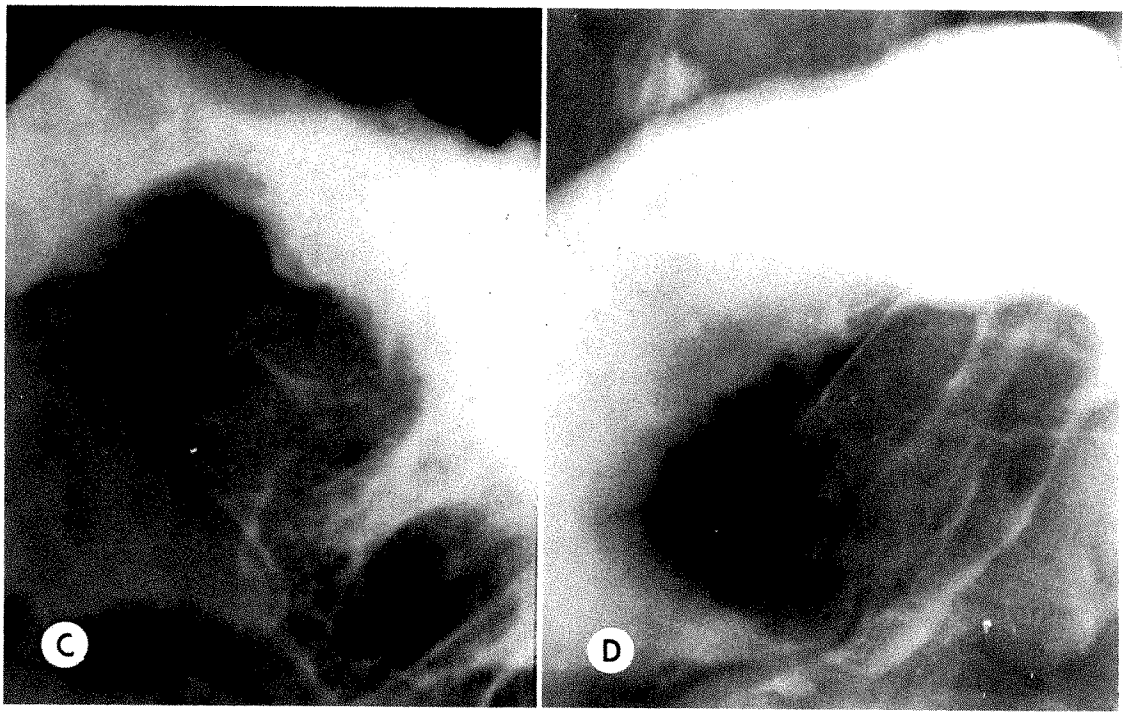


Fig. 9. (*C*) Left posterior oblique and (*D*) right posterior oblique supine spot roentgenograms. Roentgenologic diagnosis: probable ulcerating intramural tumor, possibly polypoid carcinoma.

Case III. Fig. 9, *A–F*. A 68 year old Negro male had complained of an increasingly poor appetite and a heavy sensation in the epigastrium aggravated by eating for 3 months' duration. He was examined because of a recent episode of hematemesis requiring three units of blood. Free acid was present. No malignant cells were found in gastric washings. Clinical diagnosis: carcinoma of stomach. The roentgenologic findings suggested an ulcerating intramural tumor but were not sufficiently characteristic to exclude carcinoma. Multiple determinations, which revealed no increase in phosphorus uptake over the lesion as compared to the normal antrum, suggested that the lesion was not carcinoma. A total gastrectomy was performed because at operation the lesion was thought to be carcinoma. Pathologic diagnosis: ulcerating leiomyoma.

Case IV. Fig. 10, *A* and *B*. A 53 year old white female had complaints of epigastric "aching" unrelieved by food or antacids, episodic vomiting, poor appetite and a 10 pound weight loss during the previous 6 months. On roentgenologic examination, a flattened, aperistaltic segment along the lesser curvature of the media was believed to represent infiltrating carcinoma. The differential P^{32} uptake in this area was 1.8:1. The lesion was confirmed at operation, but liver, lymph node and omental metastases were present. This case demonstrates that increased phosphorus uptake can be detected in an infiltrating, scirrhous carcinoma.

Case V. Fig. 11, *A–E*. A 70 year old white male with a history of duodenal ulcer 25 years previously, developed constant epigastric pain only partially relieved by food, progressive loss of appetite, and easy satiety during the previous 2 months. He had lost 5 pounds in weight. The clinical and roentgenologic diagnosis was carcinoma of the stomach. Differential P^{32} uptake at the margin of the ulcer was 1.25:1 suggesting that the ulcer was probably benign. Subtotal gastrectomy was performed. Pathologic diagnosis: peptic ulcer, exhibiting considerable inflammatory cell infiltration at the margins of the ulcer. In this case, as in one other benign ulcer, the slight increase in uptake was apparently related to the surrounding inflammatory reaction, but was not in the range found in neoplasm.

Case VI. Fig. 12, *A* and *B*. A 70 year old white male with severe emphysema complained of epigastric pain between meals and occasional vomiting for a period of months. Occult blood was present in the stool. Free acid was present in the stomach. Gastric exfoliative cytology was unsatisfactory. Gastroscopy was deemed inadvisable because of kyphosis. Roentgenologic diagnosis: large "indeterminate" ulcer. The P^{32} uptake ratio at the margin of the ulcer was 1.75:1 indicating neoplasm. At operation, an unresectable carcinoma was found with omental and lymph node metastases.

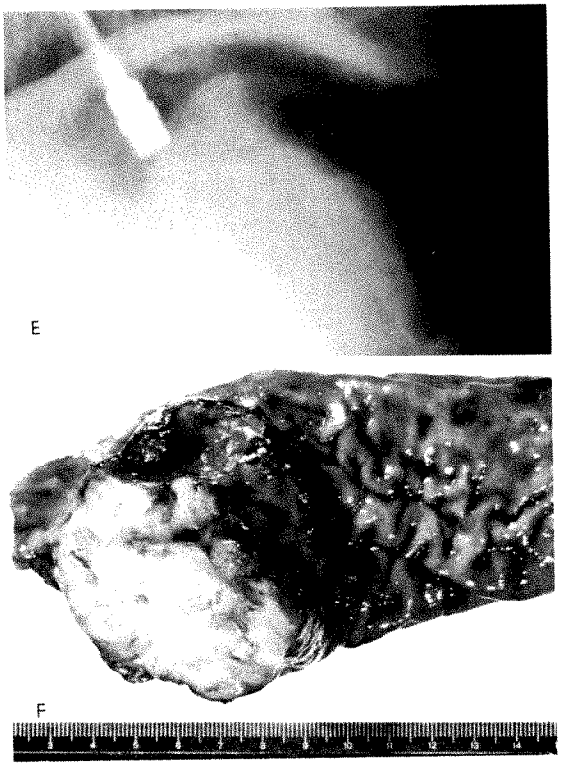


Fig. 9. (*E*) Left posterior oblique erect spot roentgenogram. The tumor is clearly outlined protruding into the air-distended stomach with the detector against it. No increase in uptake over lesion. (*F*) Photograph of resected stomach which at surgery was thought to be a carcinoma. The lesion originates in the wall. The mucosa over the surface is ulcerated. Histologic diagnosis: leiomyoma.

DISCUSSION

The results in this limited number of cases suggest that if the lesion can be accurately counted, there is excellent correlation with the uptake of the lesion and its

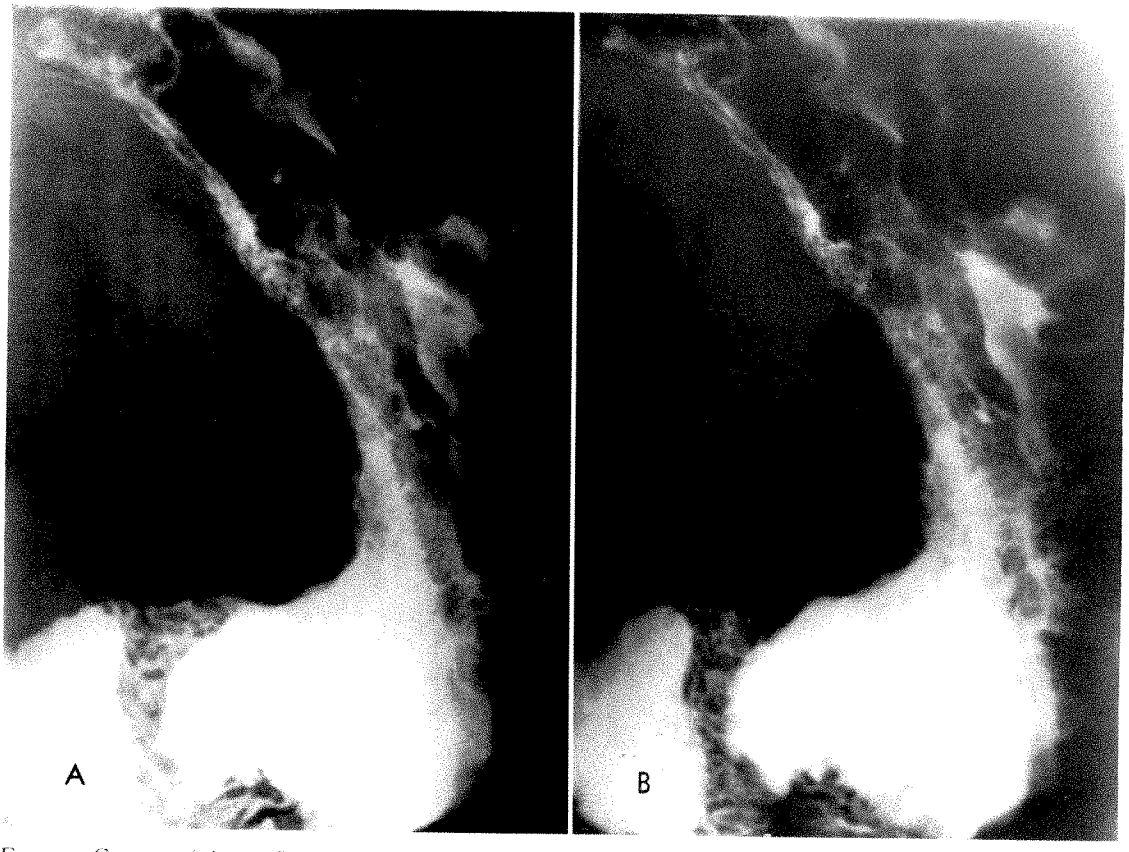


FIG. 10. Case IV. (*A* and *B*) Flattened aperistaltic area on vertical segment of lesser curve of media. No ulceration or mass. Differential uptake of this area compared to normal fundus, 1.8:1. Operation: infiltrating neoplasm of stomach metastatic to liver.

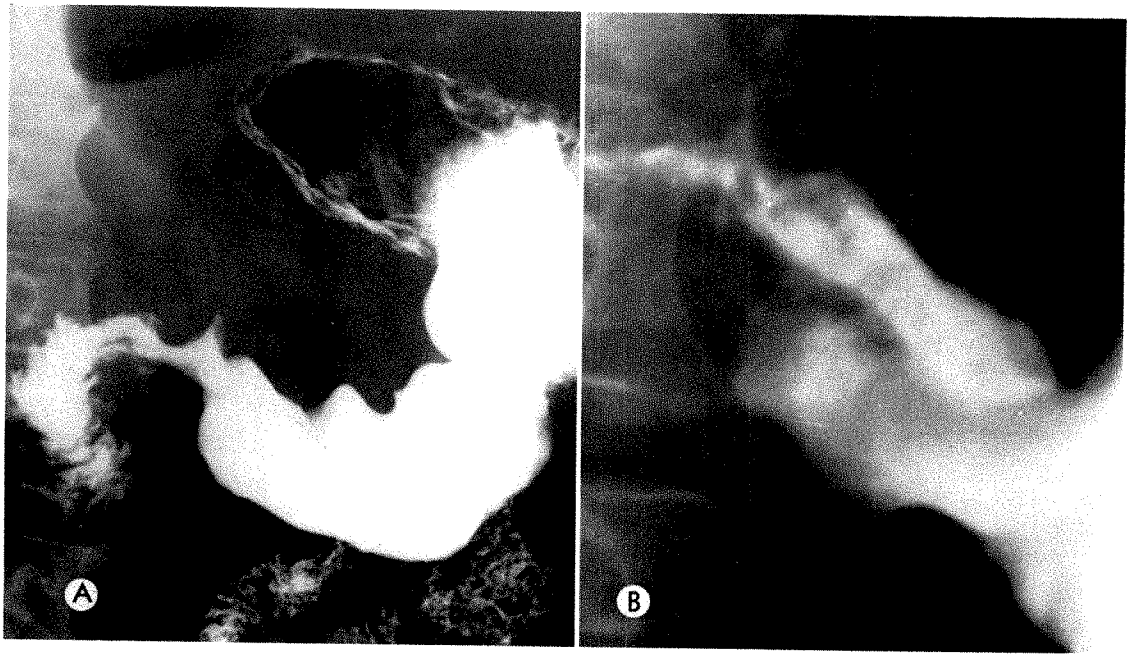


FIG. 11. Case V. (*A*) A large shallow ulcer is present on the lesser curvature in the prepyloric antrum, which on the compression spot roentgenogram (*B*) has an irregular radiolucent halo around its orifice, thought to represent carcinoma. The duodenal bulb is deformed.

histologic nature. Benign lesions showed little or no increased radioactive uptake. Of the 2 cases of peptic ulcer with differential ratios of 1.2:1 and 1.25:1, both demonstrated considerable inflammatory changes histologically. Presumably, the increased cellular activity in inflammatory tissue accounted for the slightly increased uptake in these 2 cases. In all patients with carcinoma of the stomach, the differential ratio was at least 1.5 to 1 or greater. The variability in counting rate is probably a reflection of such factors as the degree of tumor differentiation, and the presence of areas of necrosis and fibrosis.

The one false positive occurred in an elderly male with vague epigastric distress immediately after eating. A narrowed antrum which never fully dilated was noted along with a persistently enlarged rugal fold along the greater curvature. In this patient the count rate in the antrum as compared to the fundus was 2.0:1, which was thought to indicate the presence of a carcinoma. At surgery, with the stomach opened, the antrum was pliable and soft although an enlarged rugal fold was noted. A full thickness biopsy of the stomach at this point revealed normal gastric mucosa histologically, but with dilated lymph channels in the submucosa. Whether the accumulation of radioactive phosphorus in the areas of lymph stasis was sufficient to account for the increased uptake is uncertain.

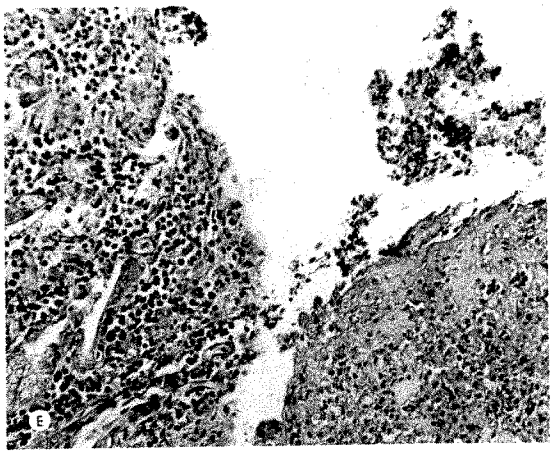


FIG. 11. (*E*) Microscopic section through edge of benign ulcer and normal mucosa (90×). Note the heavy inflammatory cell infiltration.





FIG. 11. (*C*) Autoradiograph of resected specimen. There is a slight increase in radioactivity around the margin of the ulcer. The differential uptake was 1.25:1. (*D*) Photograph of gross specimen.

The one false negative occurred in a patient with a small penetrating gastric ulcer high on the posterior wall of the lesser curvature which proved histologically to be a carcinomatous ulcer. The lesion was in an area difficult to reach with the detector, and at the time of the examination it was not certain that the lesion had been counted. Usually, the detector could be placed against the lesion during fluoroscopy. No difficulty was encountered in positioning the probe against lesions located in the antrum, fundus, or on the anterior or posterior wall of the media along the greater curvature. In this patient, and two

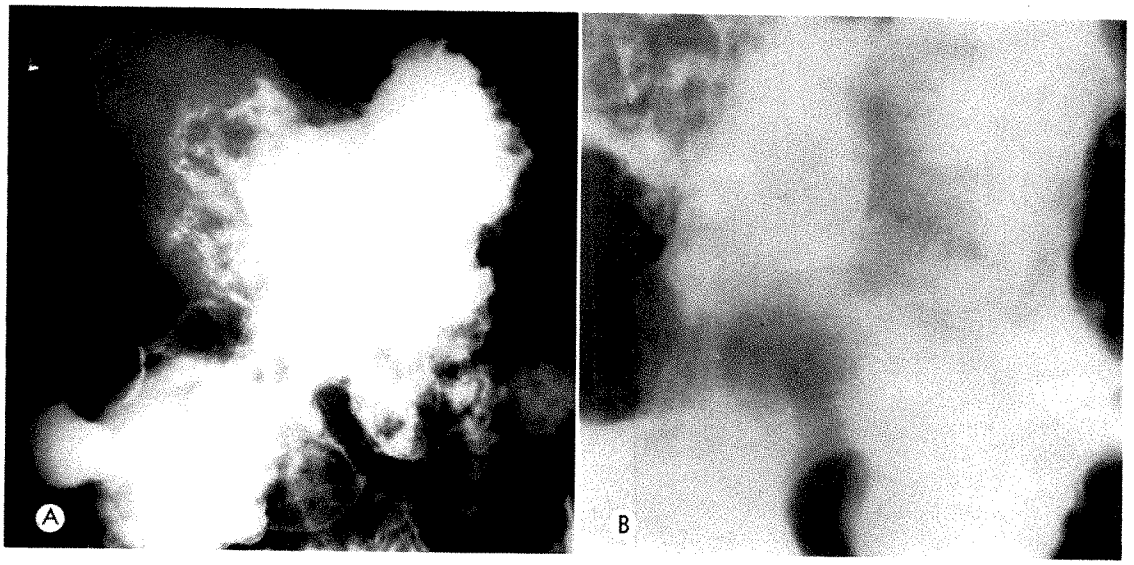


FIG. 12. Case VI. (*A*) A large penetrating ulcer on the anterior wall in a patient with advanced emphysema is best seen in the right posterior oblique view. (*B*) Spot roentgenogram shows apparently normal, though enlarged, folds up to the edge of the ulcer. Roentgenologic diagnosis: "indeterminate" ulcer. Differential uptake of 1.75:1 indicated malignancy. Inoperable carcinoma was found with omental metastases.

others with ulcers located on the posterior wall of the lesser curvature between the incisura angularis and the cardioesophageal junction, considerable difficulty was experienced in approximating the detector to the lesion. Depending on the shape of the stomach, this area may be relatively "blind" to the fluoroscopic placement technique, but is usually readily reached through the gastroscope.

While carefully performed roentgenologic and gastroscopic examination will correctly diagnose a high percentage of gastric lesions, each method has well recognized limitations. There remain many problems which neither method can satisfactorily resolve. The technique described here, supplementing conventional barium studies and gastroscopy, is safe, and easily and rapidly performed. Although the conclusions drawn must be considered preliminary, the results to date are promising and suggest that reliable information can be obtained by this simple approach which can be an extremely valuable adjunct in the clinical management of a patient with a gastric lesion.

SUMMARY

1. Preoperative gastric scans and measurement of NaI^{131} radioactivity in gastric secretions in 12 adenocarcinomas failed to reveal either selective uptake or a diagnostic pattern of secretion of the isotope. We conclude that this approach has no clinical value in the differential diagnosis of benign and malignant lesions of the stomach.

2. The physical principles of the solid state detector are briefly discussed.

3. A safe and easily performed method for detecting the selective uptake of radioactive phosphorus by gastric carcinoma using the solid state detector is described. This simple technique can reliably differentiate benign and malignant lesions of the stomach and can be a valuable adjunct to conventional roentgen studies and gastroscopy.

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THE DIAGNOSIS OF HYPERTROPHIC PYLORIC STENOSIS

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SOME physicians feel that there is little difficulty in diagnosing hypertrophic pyloric stenosis, that roentgenologic examination is not usually indicated, and that such examination may even be contraindicated.[4,8,13] On the other hand, Malmberg[10] states "examination for a palpable or nonpalpable pyloric tumor is completely unnecessary, unreliable, time consuming for the physician and unpleasant for the patient; whereas an x-ray examination gives the desired information rapidly, indisputably, and conveniently." Schaefer and Erbes[18] believe that roentgen examination is too seldom used, and that it is the most important procedure in the diagnosis of pyloric stenosis. This opinion is shared by Hefke.[9]

The true answer probably lies somewhere between these two views. The purpose of this paper is to present an analysis of cases from teaching and private hospitals where there has been no set policy pertaining to diagnosis and management of this disease.

MATERIAL

A total of 217 patients with hypertrophic pyloric stenosis has been studied. These cases were obtained from one university and three private hospitals. We were interested in (a) the presence or absence of a palpable tumor, (b) false positive tumors, (c) observation of gastric peristaltic waves, and (d) the adequacy of the roentgenologic examination.

PYLORIC TUMOR

The unequivocal presence of a palpable pyloric tumor was found in 43 per cent of all cases. Where there was disagreement between observers, the case was listed as equivocal (Table I). If all equivocal cases are counted as positive, this would bring the number of palpable pyloric tumors to 145, or 67 per cent. Thirty-three per cent had no palpable tumor; of this group 15 per cent did not have roentgenologic examination.

TABLE I

FINDINGS BY PALPATION

	No. of Cases	Per Cent
Pyloric tumor palpable	93	43
Pyloric tumor not palpable	72	33
Tumor equivocal	52	24

A striking difference in the number of palpable pyloric tumors was found by comparing the group that did with the one that did not have roentgen examination (Table II). No significant difference was found in the percentage of palpable tumors between the university and the three private hospi-

TABLE II

COMPARISON OF INCIDENCE OF PALPABLE PYLORIC TUMOR OF PATIENTS WITH AND WITHOUT ROENTGEN EXAMINATIONS

	No. with Papable Tumor (per cent)	No. with Non-palpable Tumor (per cent)	No. with Equivocal Tumor (per cent)
Patients with roentgen examination	24	54	22
Patients without roentgen examination	52	23	25

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† St. Anthony's Hospital, Oklahoma City, Oklahoma.

tal patients, being 47 per cent and 45 per cent, respectively.

A valid comparison of the number of tumors found by palpation between the surgeon and pediatrician could not be made in most instances because the diagnosis had already been established by the time of surgical consultation. However, in 57 cases a comparison could be made, and there was no significant difference.

The discrepancy in the percentage of tumors found by palpation as reported in the literature is shown in Table III.

FALSE POSITIVE TUMORS

Those cases suspected of having pyloric stenosis and a palpable tumor but with no roentgenologic or surgical evidence of the disease were called false positive. A total of 21 such patients was found in our material. This in no way reflects the true incidence since there was no coding used. Eleven of the 21 patients were operated upon and none had pyloric stenosis. Table IV is a summary of the surgical findings in these cases. The 5 patients with normal findings did not have roentgenologic examination. The other 6 patients were examined by roentgen rays and the pathology was demonstrated. The remaining 10 patients had gastrointestinal series which showed normal findings except for 3 cases with duodenal bands and malrotation.

Table III

INCIDENCE OF PALPABLE PYLORIC TUMOR AS REPORTED IN THE LITERATURE

Reference	No. of Cases	No. with Palpable Tumor (per cent)
Grimes *et al.*[7]	119	68
Rheinlander and Swenson[15]	100	90
Rinvik[16]	137	72
Savage[17]	61	80
Craig[3]		98
Miller and Friesen[12]	80	80
Gross[8]		95
Pollock *et al.*[14]	312	95
Brown[2]		44
Schaefer and Erbes[18]		63

Table IV

SURGICAL FINDINGS IN FALSE POSITIVE PYLORIC TUMORS

	No. of Cases
Malrotation with duodenal bands	4
Duplication cyst of stomach	1
Ectopic pancreas of stomach	1
Normal	5

Craig[3] feels that false positive tumors are more than just human error. He reported a series of 21 patients with vomiting of 48 hours' duration and a control group of 115 healthy infants with no vomiting. A tumor was palpated in 17 of the 21 symptomatic cases. Management limited to gastric lavage resulted in complete disappearance of symptoms in 14 of these 17 cases within 7 to 14 days. The remaining 3 were operated upon and "doubts were felt concerning the exact significance of the tumors." In the group of 115 asymptomatic newborn infants, a tumor was palpable in 25 (22 per cent).

The etiology of these false positive tumors is not known. Some are due to human error, some to palpation of other anatomic structures, and the remaining may be accounted for on the basis of pylorospasm, as suggested by Astley.[1]

PERISTALTIC WAVES

The observation of gastric peristaltic waves passing across the upper abdomen is a classic physical finding in pyloric stenosis. The incidence of this observation in the 217 cases is shown in Table v.

False positive peristaltic waves occurred in 17 of the 21 cases with false positive pyloric tumors. In Craig's[3] study peristaltic waves were present in all of the symptomatic infants with false positive pyloric tumors. Of the 115 asymptomatic infants, peristaltic waves were present in 55 or 48 per cent.

TABLE V

INCIDENCE OF PERISTALTIC WAVES OBSERVED ON PHYSICAL EXAMINATION IN 217 CASES

	No. of Cases	Per Cent
Peristaltic waves observed	100	47
Peristaltic waves absent	90	41
Peristaltic waves equivocal	27	12
Total	217	100

ADEQUACY OF ROENTGENOLOGIC EXAMINATION

Thirty-one of the 217 patients had scout roentgenograms of the abdomen. Seventy patients had gastrointestinal series with opaque material.

The scout roentgenograms were analyzed from the standpoint of (a) gastric dilatation and (b) absent gas pattern beyond the stomach. Gastric dilatation was present in 7 of 31 patients. The absent gas pattern was observed in only 4 patients, all of whom showed dehydration, supporting Feinberg and co-workers' belief that the deficiency of bowel gas is largely due to dehydration and not pyloric obstruction.[6] A normal gas pattern was seen in 23 other patients who also had significant dehydration.

Of the 70 infants who had studies with opaque material, barium was used in 63 cases, lipiodol in 4, and aqueous opaque media in 3. The results are shown in Table VI.

True gastric dilatation is uncommon in pyloric stenosis, probably much less than the 19 per cent recorded in Table VI. This may be due to the fact that the obstruction is relatively acute. Gastric distention was common in infants with pyloric stenosis, but disappeared after "burping" as it does in normal infants.

TABLE VI

FINDINGS IN 70 CASES WHICH HAD GASTROINTESTINAL ROENTGEN EXAMINATION

	No. of Cases	Per Cent
Gastric Dilatation	13	19
Esophageal Reflux	48	69
Beak Sign	34	49
String Sign	59	84
Tit Sign	62	89

The presence of esophageal reflux and a pyloric "beak" are directly related to the diligence of search. Reflux was observed and recorded five times as frequently in those patients examined during the period 1957–1962, as compared to the 1952–1957 period. The "beak" should be seen in all cases since it merely represents barium in the proximal portion of the stenosis (Fig. 1).

The "string" sign as first described by Meuwissen and Slooff[11] actually represents barium in the narrowed lumen, surrounded by tumor. It is the most reliable sign of pyloric stenosis, being seen in no other condition (Fig. 2).

The pyloric "tit" is a newly described finding in pyloric stenosis.[19] It is thought to be due to a persisting peristaltic wave which cannot pass downwards because of the pyloric tumor (Fig. 3).

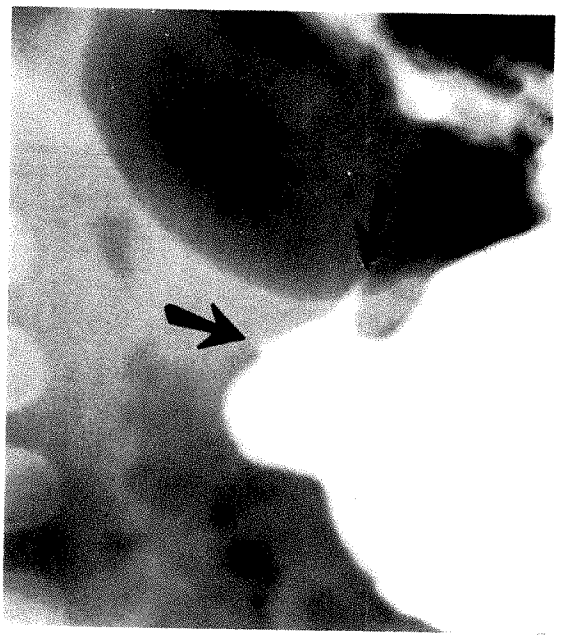


FIG. 1. The pyloric "beak" is indicated by the lower arrow and the pyloric "tit" by the upper one. The "beak" is formed by barium entering the proximal portion of the narrow pyloric canal.

The roentgen examination was considered satisfactory if a pyloric "string" was demonstrated. There were 11 unsatisfactory examinations. Of these 11 patients, only 1 had a palpable pyloric tumor, and 3 had equivocal palpable tumors. Four of these unsatisfactory examinations were performed with lipiodol, 3 with aqueous opaque medium and 3 with barium.

There were no false negative or false positive examinations. In 1 patient an upper gastrointestinal series 1 week prior to transfer to the University Hospital was reported as showing an anomaly, but no pyloric stenosis.

The only complication of roentgen examination was aspiration of the opaque medium in 5 patients. Three of these were examined with lipiodol, 1 with barium, and 1 with an aqueous opaque medium. In our experience barium sulfate remains the superior medium, yielding a significantly higher percentage of satisfactory studies. We find that the incidence of aspiration is low when the examination is properly done.



Fig. 2. The "string" sign is shown by the lower arrow. At the left of the "string" is the duodenal bulb showing the mushroom impression of the pyloric tumor in its base. Proximal to the "string" sign is the pyloric "tit" (upper arrow).



Fig. 3. The pyloric "tit" (arrow) is a segment of the antral wall that is squeezed against the pyloric tumor by a peristaltic wave. It is the first indication of pyloric obstruction seen on fluoroscopic examination.

Barium aspiration is no worse than aspiration of any other opaque agent according to Dunbar *et al.*[5]

DISCUSSION

The finding of an unequivocally palpable tumor in this group of 217 patients with pyloric stenosis was low. The 43 per cent incidence is thought to be representative of the general situation as the patients included were seen by numerous physicians with different methods of examination and no set policies as to diagnosis and management. Undoubtedly, palpable pyloric tumors would be found more often if the patients were examined in hospitals with a "no palpable tumor—no surgery" policy. Contrariwise, the incidence of the pyloric tumors would be even lower than noted above if a policy of "roentgenologic examination in all cases irrespective of the presence of a tumor" would be followed. The fact remains that a palpable pyloric tumor was not a reliable sign in the diagnosis of pyloric stenosis, particularly when the false positives are considered.

Roentgenologic studies in this group of

patients were likewise not without failure; however, a diagnostic accuracy of 84 per cent was obtained, and there is good evidence to indicate that this will be improved. For example, of the 11 unsatisfactory examinations, 8 were performed prior to 1957, and the remaining 3 were done using an aqueous opaque medium. Also, the improved accuracy of the roentgen examinations is reflected in the fact that prior to 1957 only 20 per cent of the patients had roentgenographic studies, whereas 70 per cent had such examinations since 1957. We found no evidence to support the view that the roentgenologic examination is harmful or interferes with the management of the patient.

CONCLUSIONS

1. The palpation of a pyloric tumor was found to be an unreliable sign in the diagnosis of pyloric stenosis in a significant series representing good pediatric and surgical practice.

2. The roentgenologic examination is accurate, reliable, and has a low incidence of complications when properly done.

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EVALUATION OF CONVENTIONAL ROENTGENOGRAPHIC TECHNIQUES FOR DEMONSTRATION OF DUODENAL ULCER CRATERS*

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DUODENAL ulcer is a common lesion. According to Bockus,[3] 10 per cent of the male population in this country suffers from peptic ulcers, and 66 per cent to 75 per cent of these are in the duodenum. Since most of the patients with duodenal ulcer do not die or undergo surgery and since the history and physical examination may be suggestive of peptic ulcer but the symptoms might actually be due to some other cause, the radiologist must be relied upon for the accurate clinical diagnosis of the majority of duodenal ulcers. With the advent of newer techniques (image intensification fluoroscopy and cinefluorography), one cannot help but wonder as to the future of older methods for the detection of duodenal ulcer by currently conventional fluoroscopy and roentgenographic study. With this in mind, it was considered desirable to evaluate our methods for the detection of duodenal ulcer craters and to determine which parts of the examination were most fruitful and which were the least accurate.

The standard textbooks[5,8,12,13,14,15] in diagnostic roentgenology emphasize fluoroscopy and compression spot roentgenography of the duodenal bulb in the erect right anterior oblique position and the left anterior (reverse) oblique position. The routine use of multiple Bucky exposures of the duodenal bulb in the prone right anterior oblique position with compression has not been emphasized. Supine, right side elevated, barium-air contrast spot roentgenography has not been employed except by Hampton,[6] Hinkel and Moller,[7] and Chucker and Finestone,[4] who urge the routine use of the supine barium-gas* contrast study of the duodenal bulb.

Meyer[9] compared the use of the barium-air contrast spot roentgenograms with erect compression roentgenograms in consecutive patients and found the crater demonstrated only by the barium-air contrast spot roentgenograms in 8 per cent of 176 instances. Hinkel and Moller[7] evaluated 50 consecutive patients in whom a crater was demonstrated and found that the crater was visualized only on the "compression" (position of patient not specified) roentgenograms in 5 per cent, only on barium-gas contrast spot roentgenograms in 10 per cent, and on both, the compression roentgenograms and barium-gas spot roentgenograms in 85 per cent.

METHOD

In order to assess the value of the various projections described for the demonstration of duodenal ulcer, and to compare the results of fluoroscopic observations with conventional roentgenographic examinations, the following study was undertaken.

One hundred and ten duodenal ulcer craters in 107 consecutive patients in which the roentgenographic study was considered adequate were reviewed by the authors. No case was included unless it was considered unequivocally diagnostic of an ulcer crater by all of the reviewers. Each examination consisted of a careful fluoroscopic study of the stomach and duodenum in the erect,

* Chucker and Finestone and Hinkel and Moller utilized carbon dioxide gas from carbonated beverages or other means to distend the barium-coated bulb rather than depending on swallowed air, but the roentgenographic result is essentially the same.

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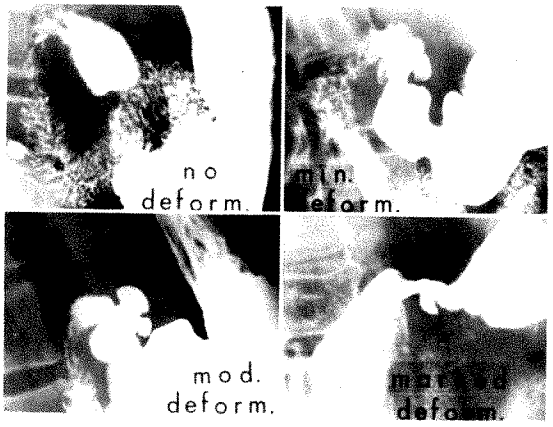


FIG. 1. Criteria of deformity demonstrated on roentgenograms of 4 different patients. *Upper left:* normal. *Upper right:* minimal deformity. *Lower left:* moderate deformity. *Lower right:* marked deformity.

supine and prone positions as well as a fluoroscopically controlled routine but detailed roentgenographic study. The roentgenographic part consisted of the following: (1) erect spot roentgenograms of the bulb (4 exposures on one 8×10 inch film) with compression in the right anterior oblique position, (2) a supine air-contrast spot roentgenogram (4 exposures on one 8×10 inch film) with the right side elevated, (3) a prone oblique serialogram (4 Bucky exposures on one 10×12 inch film) with the left side elevated, and (4) a prone oblique serialogram with compression. Compression in the prone oblique position was accomplished by means of a pneumatic paddle[1,2] permitting varying degrees of pressure. In each position the bulb was rotated into the longest profile possible, in an attempt to free it from the superimposed shadow of the stomach or duodenal loop. Compression of the bulb was varied in the erect and prone positions until its mucosal pattern was demonstrated. When necessary, several sets of roentgenograms were made in an effort to demonstrate the mucosal pattern of the entire bulb adequately.

The diagnosis of a crater was made when a constant, unchanging collection or projection of barium was seen on all 4 roentgenograms made in the position being evaluated. Each position was evaluated as to whether the crater was demonstrated unequivocally, whether there was an equivocal demonstration, or whether the crater was not shown at all. An equivocal rating was given when the reviewers did not think a collection or projection of barium was noted with sufficient clarity or constancy on the 4 roentgenograms of a given position to permit the diagnosis of a crater.

In addition, deformity of the bulb was graded as none, minimal, moderate, or marked (Fig. 1). Irritability of the cap was based on finding frequent incomplete filling of the bulb on the roentgenogram in spite of a normal rate of gastric emptying and barium in the loop. The fluoroscopist's notes as to detection of a crater, point tenderness, or irritability of the bulb were also recorded.

The location and average diameter of each crater was recorded and the size of the crater was correlated with fluoroscopic detection. It is of interest to note that 53 per cent of the ulcers were located in the proximal third of the cap (Fig. 2), indicating the need for adequate visualization of the base of the bulb by compression and air-contrast.

Some radiologists omit the supine air-contrast spot and prone compression roentgenograms and evaluate the duodenum only as visualized on large roentgenograms exposed primarily for the stomach in various positions by the technician without

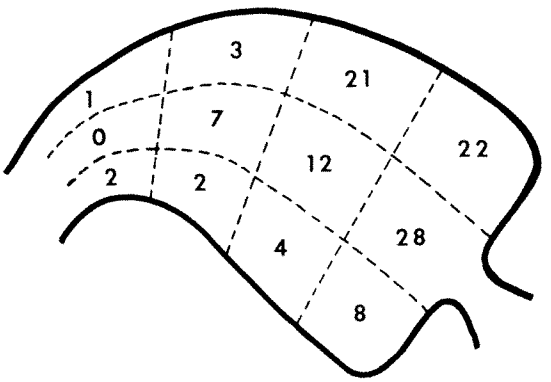


FIG. 2. Diagram showing distribution of ulcer craters in the bulb and postbulbar region as found in an analysis of 110 instances among 107 patients.

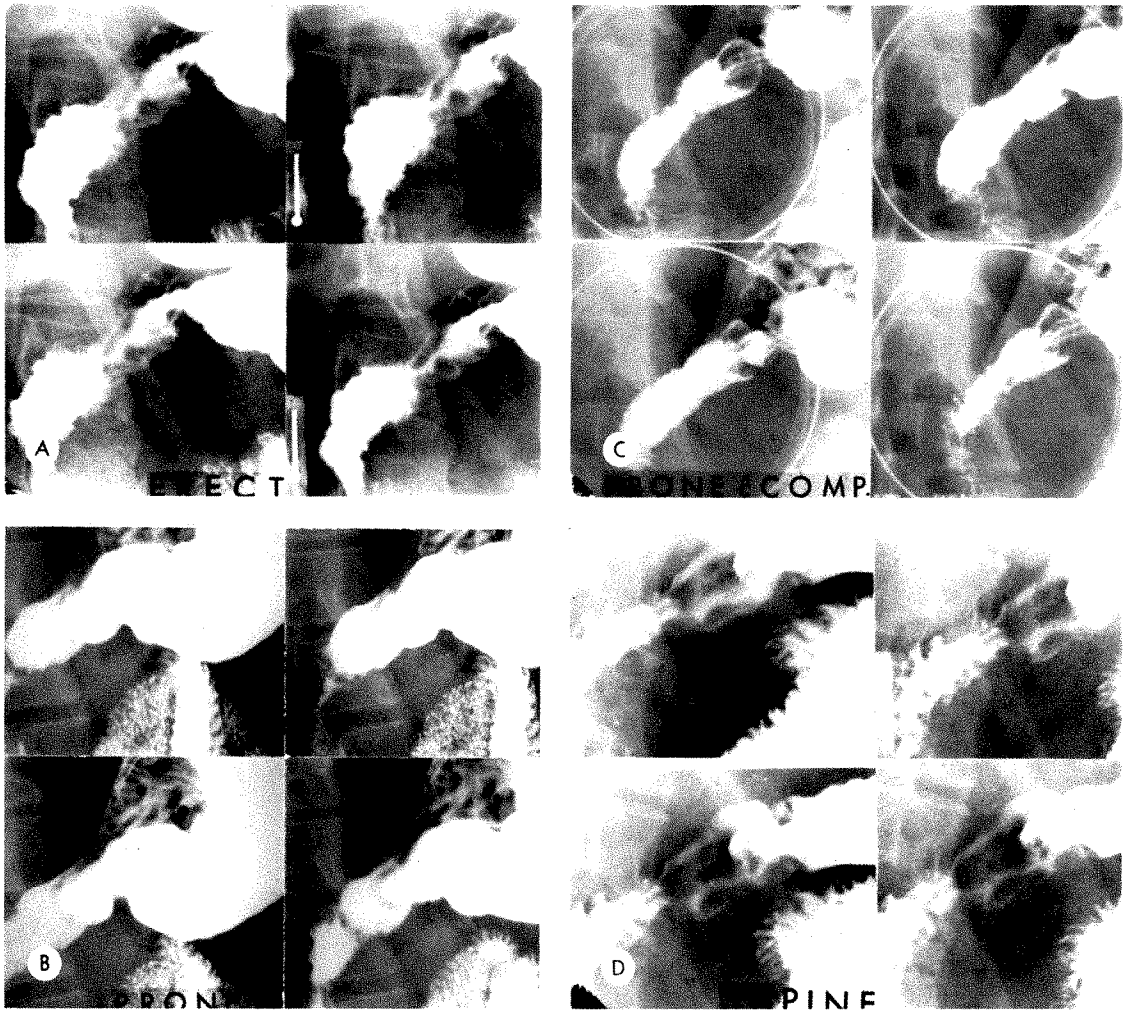


Fig. 3. Four different views of the duodenal bulb of the same patient. (*A*) Set of erect spot roentgenograms (4 on one 8×10 inch film), (*B*) prone serialograms without compression (4 on one 10×12 inch film), and (*C*) prone serialograms with compression. No crater is seen on any of these roentgenograms. (*D*) Set of supine air-contrast spot roentgenograms demonstrating ulcer crater.

benefit of fluoroscopic control to secure optimum positioning. Therefore, as part of this study we evaluated the accuracy of the diagnosis of duodenal ulcer from our routine roentgenograms of the stomach. These include 10×12 inch films of the stomach and duodenum exposed by the fluoroscopist for each patient in the erect right anterior oblique, supine right side elevated, and prone oblique left side elevated projections. In this study, the films exposed primarily for the stomach were considered positive for duodenal ulcer if a crater was either equivocally or unequivocally demonstrated. A crater was considered equivocally demonstrated if it was sufficiently definite to alert a reviewer to secure a more detailed examination of the bulb.

RESULTS

The supine barium-air contrast spot roentgenograms of the bulb proved to be the most valuable, demonstrating 75 per cent of the craters. Twenty-four and one-half per cent of the craters could be diagnosed only in these exposures (Tables 1 and 11; Fig. 3, *A–D*). Since the supine position is

TABLE I

DEMONSTRATION OF 110 DUODENAL CRATERS IN VARIOUS POSITIONS

Position	Not Seen	Equivocal	Diagnostic
Supine Air Contrast	15 (14%)	12 (11%)	83 (75%)
Prone Oblique Compression	25 (23%)	15 (14%)	70 (63%)
Erect	41 (37%)	23 (21%)	46 (42%)
Prone Oblique, NoCompression	61 (56%)	15 (14%)	34 (30%)

17.3% of craters were demonstrated in all positions.

optimal for demonstrating posterior wall craters, the above findings are in accord with autopsy statistics which show a preponderance of posterior wall craters (59.4 per cent,[10] and 61.9 per cent[11]).

Prone oblique serialography with compression proved to be the next most efficient technique, demonstrating 63 per cent of the craters and was the only diagnostic method in 12.5 per cent (Fig. 4).

The erect right anterior oblique position was of value in demonstrating 42 per cent of the craters and was the only diagnostic projection for demonstrating 2 craters (1.8 per cent) (Fig. 5).

The prone oblique serialograms without compression were diagnostic in 30 per cent of the cases but did not demonstrate any crater that was not demonstrated in the prone oblique serialogram with compression. Although these studies were the least

TABLE II

DUODENAL CRATERS DEMONSTRATED IN ONE POSITION ONLY

Position	No. of Craters	Per Cent of 110 Craters
Supine Air Contrast	27	24.5
Prone Oblique Compression	14	12.5
Erect	2	1.8
Prone Oblique, No Compression	0	0

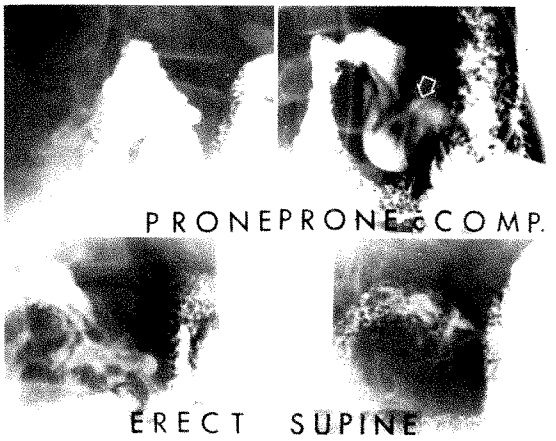


FIG. 4. One roentgenogram from each set of exposures made of same patient in various positions; crater (arrow) demonstrated only in prone serialogram with compression.

valuable for demonstrating craters, they were the most important for evaluation of deformity and evidence of extrinsic pressure on the bulb or proximal duodenal loop.

The erect left anterior (reverse) oblique roentgenograms did not, in this series, demonstrate any craters which were not demonstrated, at least equivocally, in some other projection. Nevertheless, in our larger experience, we think that it is a useful projection since occasionally it has permitted a definite diagnosis of ulcer crater which could not have been made with certainty in

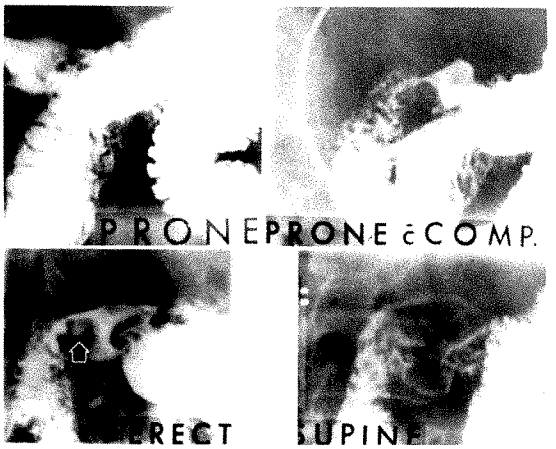


FIG. 5. One roentgenogram from each set of exposures made of same patient in various positions; crater (arrow) demonstrated only in erect position.

any other projection. Roentgenograms in this projection are not routine in our department and often cannot be obtained because of the habitus of the patient. Reverse oblique roentgenograms are usually made only when the cap cannot be visualized in its entirety without being obscured by the superimposed gastric antrum or duodenal loop. These roentgenograms were available in only 23 instances and demonstrated 14 (60 per cent) craters.

Forty-one per cent of the craters were seen both in the prone oblique serialogram with compression and in the supine barium-air contrast spot roentgenograms.

Deformity was absent in 13.6 per cent of 107 cases, and was minimal in 37.3 per cent (Table III). The importance of a careful study of the undeformed bulb as well as the deformed bulb is obvious. Templeton[15] found no deformity in 18.4 per cent of 283 cases in which a crater was demonstrated.

The so-called secondary signs of duodenal ulcer were quite unreliable, irritability having been noted in only 8 instances (7.3 per cent) and tenderness was present in 6 patients (5.5 per cent) in this series.

Although much emphasis has been placed on fluoroscopy by many radiologists, its reliability in this study was poor. Fifty-seven (52 per cent) of 110 craters were visualized by the fluoroscopist. The most experienced radiologists, each of whom has had more than 10 years' experience, were able to detect less than 70 per cent of the craters (Table IV). As might be expected, less experienced fluoroscopists detected fewer craters.

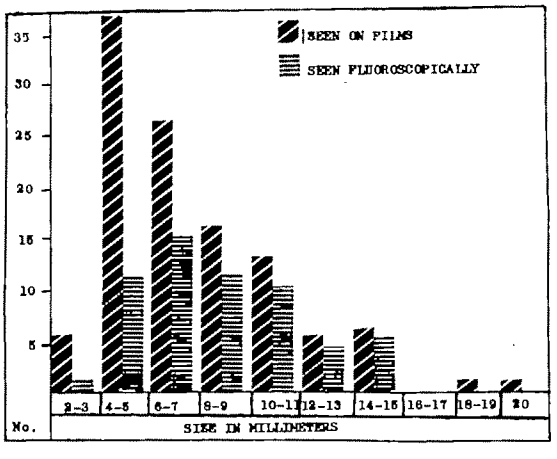


FIG. 6. Relationship of fluoroscopic detection to crater size.

TABLE III

DEFORMITY OF DUODENUM ASSOCIATED WITH 110 ULCER CRATERS

Deformity	No.	Per Cent
None	15	13.6
Minimal	41	37.3
Moderate	39	35.5
Marked	15	13.6

The average diameter of the craters is shown in relation to detection at fluoroscopy in Figure 6. Approximately two-thirds of the craters less than 6 mm. in diameter were not diagnosed at fluoroscopy. The location of the craters is given in Figure 2, and corresponds fairly well with reports by others.[14,15]

Fifty-nine per cent of the craters were undetected either on the erect, supine or prone oblique roentgenograms made primarily for the stomach but which also included the bulb.

DISCUSSION

The roentgen diagnosis of duodenal ulcer depends on the demonstration of the ulcer crater. For many years the 4 positions described in this paper have been used in our department in the routine examination of the duodenum, yet no one, to our knowledge, has attempted to assess the relative value of these positions for the demonstration of ulcer craters.

TABLE IV

FLUOROSCOPIC ACCURACY IN 110 DUODENAL CRATERS

Fluoroscopist	No.		Per Cent
	Seen	Cases	
Residents	31	67	46
Staff	26	43	61
Senior Staff #1	15	22	68
Senior Staff #2	9	13	69

If reliance is placed solely on fluoroscopic observations, our results indicate that almost one-third of the demonstrable craters *would not* be diagnosed by experienced fluoroscopists and that more than half the ulcers would be missed by less experienced observers. These observations confirm the opinion of others regarding the inadequacy of conventional fluoroscopy.

Almost 60 per cent of the ulcer craters were not demonstrated on the ordinary roentgenograms of the stomach and duodenum made in the erect, prone oblique and supine oblique positions.

If one limits the roentgenographic study of the cap to erect spot roentgenograms, our experience indicates that only 42 per cent of the demonstrable craters will be identified, and of these, less than 2 per cent will not be adequately demonstrated in another projection.

Similarly, multiple prone roentgenograms of the cap without compression will only show 30 per cent of ulcer craters and will not show any crater that cannot be seen in some other position.

The most valuable position is the supine oblique air contrast spot exposure. In this position, 75 per cent of all the craters were demonstrated; about one-fourth of all the ulcers could be identified only in this position. The prone oblique roentgenograms of the duodenum made with compression on the cap were adequate for showing 63 per cent of the ulcers; about 12 per cent were seen only in this position. More than 98 per cent of the craters in this series were demonstrated by exposures in these two positions.

From these observations, one can conclude that no examination for peptic ulcer of the duodenum is complete without exposures of the cap made in the supine position with air-contrast and in the prone oblique position with compression that is adequate to reveal the mucosal pattern.

SUMMARY

1. The only reliable roentgen sign of duodenal ulcer is demonstration of the ulcer crater.

2. Comprehensive study of the duodenal bulb for an ulcer crater should consist of a fluoroscopically controlled detailed roentgenographic study that includes multiple exposures in the prone oblique position with adequate compression and in the supine oblique barium-air contrast projection.

3. The secondary signs of activity (irritability and tenderness) were present in only approximately 6 to 8 per cent of 107 cases in which the crater was demonstrated.

4. Deformity of the bulb in the presence of an ulcer crater was minimal or absent in 50 per cent of the cases.

5. Fluoroscopy was unreliable in the diagnosis of duodenal ulcers; the most experienced fluoroscopist detected less than 70 per cent of the craters. The over-all accuracy of fluoroscopic detection was 52 per cent.

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PLAIN ROENTGENOGRAPHIC FINDINGS IN DRUG INDUCED INTRAMURAL HEMATOMA OF THE SMALL BOWEL

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AN ALERT radiologist can diagnose or at least suspect an intramural hematoma of the small bowel on routine abdominal roentgenograms, especially if he is aware that the patient is receiving anticoagulant drugs. This is particularly true for lesions of the jejunum or ileum, there being fewer confusing gas shadows to obscure the pathology.

The purpose of the authors is to present 4 cases of intramural hematoma of the small bowel secondary to anticoagulant therapy. In 3 of these, the diagnosis was made prior to contrast studies. A diagnosis of mesenteric vascular occlusion advanced in the fourth case would have been tempered had it been known that the patient was receiving coumadin.

REVIEW OF THE LITERATURE

Intramural hematoma of the small bowel has been diagnosed frequently in recent years following the emphasis given this entity by Felson and Levin[3] in 1954. Most cases reported to date were of traumatic origin and involved the duodenum. Drug induced hematomas, on the other hand, tend to involve the jejunum or ileum. The value of barium studies in the preoperative diagnosis of hematomas secondary to anticoagulant therapy was demonstrated by the reports of Culver *et al.*,[1] Wiot *et al.*[7] and Senturia *et al.*[5] in 1961. They reported 1, 2 and 3 cases, respectively. We were able to find records of 6 other similar cases[3,6] in addition to the 4 described in this report. We have encountered another intramural hematoma of the jejunum in a hemophiliac. A volvulus was also present at surgery. In retrospect, the plain roentgenographic findings parallel those under discussion. The only cases of intramural hematoma diagnosed on routine abdominal roentgenograms prior to barium studies were those reported by Silbert *et al.*[6] We have verified their impressions in our cases and wish to emphasize the importance of the plain roentgenographic findings in the diagnosis of this disease.

REPORT OF CASES

CASE 1 (Fig. 1, *A* and *B*). A 68 year old female was admitted to the Baylor University Medical Center on July 25, 1960 with a 3 day history of abdominal pain, dysuria and hematuria. One day prior to admission, the abdominal pain became generalized and dark vomitus was noted. Bowel movements were normal. Past history revealed a previous hospitalization on June 3, 1960 for removal of a renal cyst. While hospitalized, the patient developed a pulmonary embolus and was placed on dicumarol, alternating 50–100 mg. doses daily. A prothrombin determination 10 days prior to admission was 20 per cent. Physical examination revealed a moderately dehydrated patient in pain. Positive physical findings consisted of generalized abdominal distention, rebound tenderness and slight abdominal rigidity. Prothrombin time upon admission was less than 5 per cent, the hematocrit 37 per cent, dropping 3 days later to 25 per cent. Abdominal roentgenograms upon admission were interpreted as "a possible closed loop obstruction." A barium enema study was normal. A small bowel series was reported as showing "distinctly abnormal loops of small bowel suggesting an intramural process, due either to edema or hemorrhage." Nausea and abdominal tenderness diminished over the next 5 days. Nine days after admission, exploratory surgery revealed 500 cc. of old blood in the abdominal cavity. Two segments of lower jejunum approximately 12 cm. in length were roughly 3 times the thickness of normal adjacent bowel due to hemorrhage which had extended subserosally into the root of the mesentery. The involved loops had good peristaltic

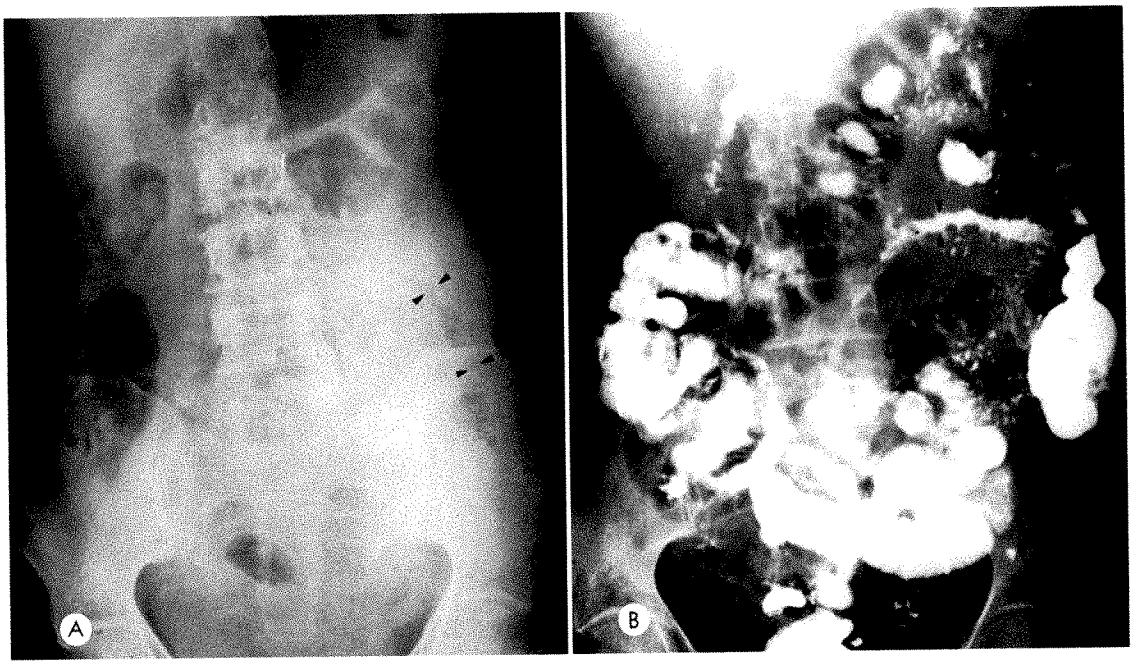


Fig. 1. Case 1. (*A*) "Thick bowel sign" demonstrated by gas within a greatly narrowed bowel lumen. The typical irregular mucosal folds are seen. The pseudotumor results from the hemorrhagic bowel. The blurring of the left psoas border is due to bleeding into the root of the mesentery. (*B*) The abnormal loop is outlined by barium. The mobility of the bowel does not appear impaired as in a pseudotumor due to an obstructing band. There is little or no evidence of small bowel obstruction.

activity and appeared viable, hence resection was declined. The patient recovered rapidly and was discharged 17 days after admission. A follow-up small bowel series 2 months later was normal.

Case II (Fig. 2, *A* and *B*). A 58 year old male was admitted to the Baylor University Medical Center on September 10, 1961 with a 5 day history of increasingly severe cramping, mid-abdominal pain and tenderness. Nausea, vomiting and anorexia presented 30 hours prior to admission. There was no history of melena or hematemesis. Pertinent past history revealed a previous hospital admission 3 months earlier for a myocardial infarction. He recovered and had been well until the present illness, being maintained on coumadin, 5 mg./day. Physical examination revealed tenderness in the right abdomen without distention. The bowel sounds were described as "loud and frequent." No other pertinent physical findings were elicited. Levin tube drainage on the day of admission consisted of dark red blood. The prothrombin time on September 11, 1961 was 10 per cent, the hematocrit 34 per cent. Urinalysis revealed numerous red blood cells. An upper gastrointestinal series with gastrografin on the same day was interpreted as "small bowel obstruction." A barium study on September 12, 1961 showed "typical findings of intramural hematoma of the small bowel." A repeat examination on September 16, 1961 showed that there was definite but not complete absorption of the hematoma. The patient was treated conservatively with vitamin K. By the third hospital day there was no evidence of gastrointestinal hemorrhage and the abdominal discomfort had improved. The patient gradually became asymptomatic and was discharged.

Case III (Fig. 3, *A* and *B*). A 67 year old female was admitted to the Baylor University Medical Center on August 1, 1962 with a 2 day history of noncramping, abdominal pain, nausea and vomiting and a 1 day history of melena. Pertinent past history revealed that the patient had been hospitalized in 1961 with a diagnosis of cerebral atherosclerosis. A daily dose of 5 mg. of coumadin, prescribed at that time, was continued up to the present admission. Physical examination revealed an acutely

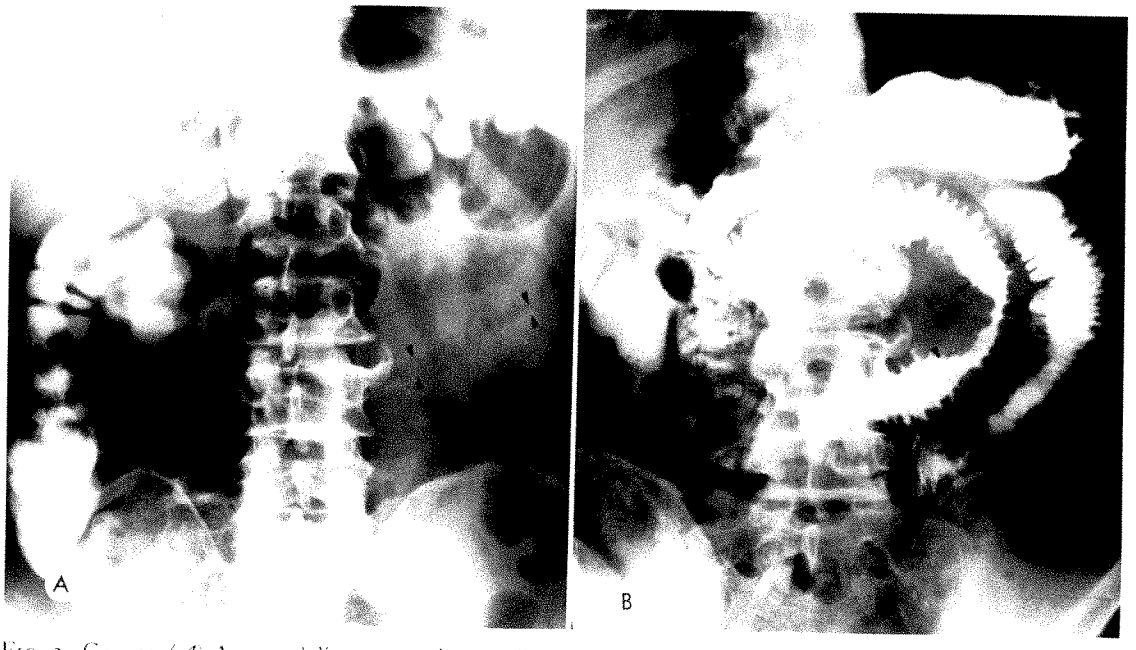


FIG. 2. Case II. (*A*) Arrows delineate an abnormally narrow, continuous gas pattern in a thickened tubular loop of small bowel. Signs of partial small bowel obstruction are evident. Gastrografin outlines the colon. (*B*) Barium study demonstrates the typical "picket fence appearance" of intramural hematoma in two adjacent loops of jejunum.

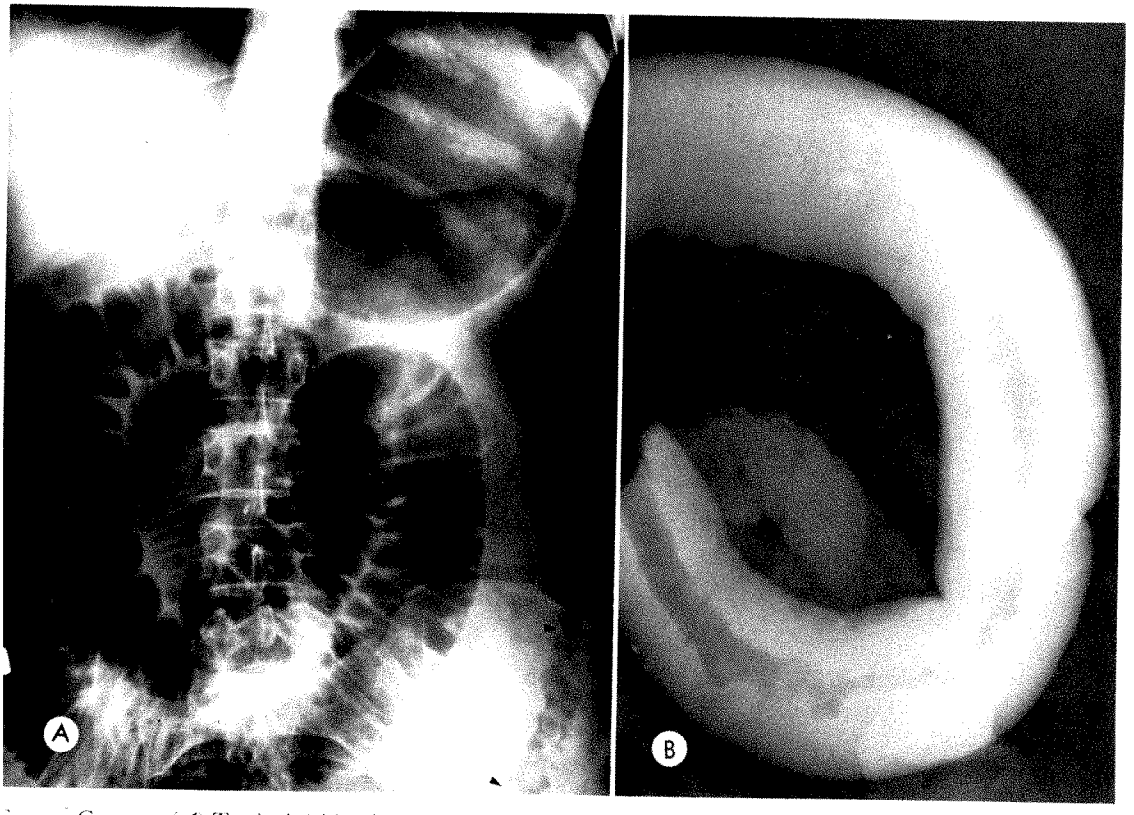


FIG. 3. Case III. (*A*) Typical "thick bowel sign" (arrows) with the proximal small bowel markedly distended, indicative of mechanical obstruction. (*B*) Pathologic specimen removed at surgery showing encroachment upon the bowel lumen due to the hemorrhage into the bowel wall.

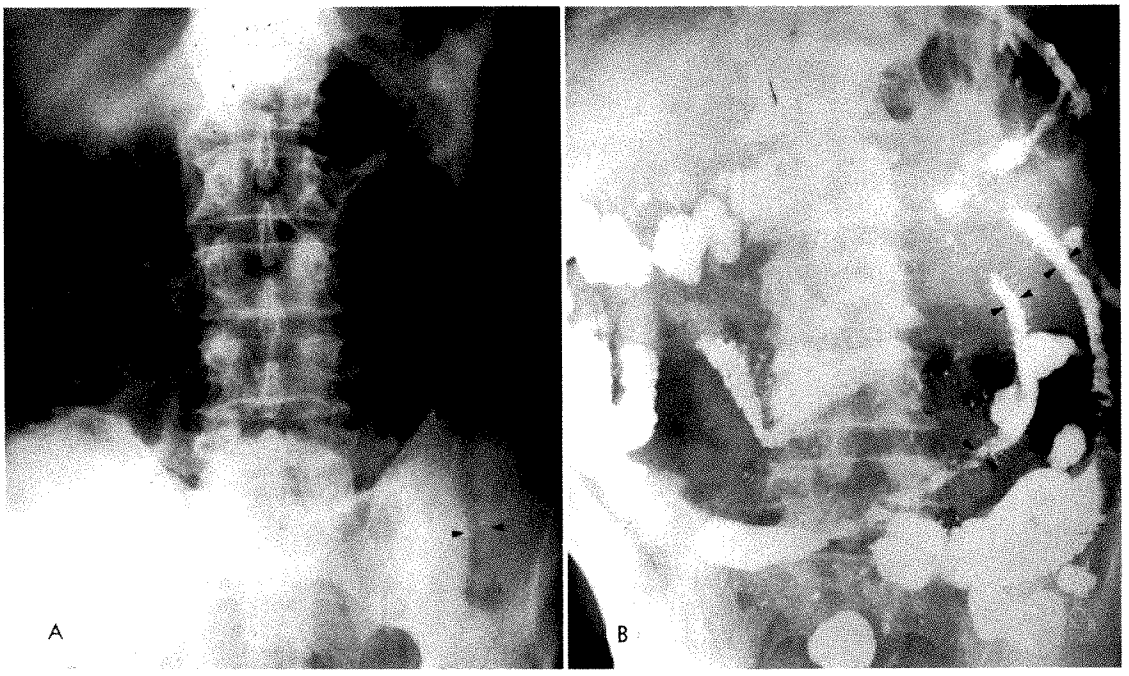


FIG. 4. Case IV. (*A*) Typical finding of an abnormal gas pattern due to bowel thickening with predominant colon distention and little or no evidence of abnormal small bowel distention. (*B*) Small bowel study demonstrating incomplete obstruction. Barium outlines the abnormal bowel lumen in the involved loops. The impression of a mass around this area is due to the hematoma and possibly to retroperitoneal bleeding.

ill female, periodically vomiting coffee ground material. Positive physical findings revealed hyperperistalsis, right lower quadrant tenderness without rebound and moderate abdominal distention. Abdominal roentgenograms on admission showed several markedly dilated loops of small bowel initially interpreted as "mechanical small bowel obstruction, possibly due to volvulus." A small, tubular gas shadow in the left lower quadrant was interpreted later as "mesenteric vascular occlusion or intramural hematoma, knowing that the patient is receiving anticoagulants." Admission prothrombin time was 8 per cent, the white blood cell count 14,200, and the hematocrit 25 per cent. After correcting the prothrombin level, a laparotomy was performed. Eighteen inches of hemorrhagic, stiffened jejunum with several areas of gangrene were removed. Mesenteric hemorrhage was also noted. Normal arterial pulsations were palpable to the bowel edge. Microscopic examination showed extensive hemorrhage into the submucosa of the bowel. The patient recovered rapidly and was discharged in good condition.

CASE IV (Fig. 4, *A* and *B*). A 66 year old male was admitted to the Baylor University Medical Center on May 5, 1963 with a 4 day history of increasingly severe, cramping abdominal pain which was generalized and did not radiate. Two years prior to admission the patient had been hospitalized with a myocardial infarction from which he made a fair recovery. Coumadin had since been prescribed in doses of 5 mg. daily. Physical examination revealed a large ecchymotic area over the right shoulder, diminished peristaltic activity, abdominal distention and tenderness. Abdominal roentgenograms on admission revealed marked colon distention to the splenic flexure and were interpreted as "possible colon obstruction." A barium enema study was normal and the roentgenologic diagnosis was changed to "probable bowel infarction or intramural hematoma." On admission the prothrombin time was 8 per cent, hematocrit 43 per cent and white blood cell count 13,400. Treatment consisted of aqueous vitamin K. On the second hospital day the patient began to improve with a decrease in bowel distention and a return of normal bowel sounds. On the sixth hospital day the patient was discharged asymptomatic.

DISCUSSION

Intramural hematoma of the small

bowel, once considered a relatively rare complication of anticoagulant therapy is reported with increasing frequency. The ability to recognize this abnormality assumes great importance because of the expanding use of anticoagulant therapy. The radiologist, aware of this iatrogenic disease and its roentgen manifestations is in a position to obviate extensive surgery on borderline patients already compromised by a defective circulatory system due to atherosclerosis.

In order to diagnose intramural hematoma from the plain roentgenograms, one should be familiar with the "thick bowel" or "crescent" sign described by Wolf *et al.*[8] and Nelson and Eggleston[4] in cases of small bowel infarction due to mesenteric vascular disease. This sign, in addition to being present in these two diseases, may also be found in tuberculosis, lymphoma and regional enteritis involving the jejunum or ileum. The sign we wish to stress consists of thin curvilinear, continuous gas shadows delineating a narrowed bowel lumen which is coarsely irregular from distorted mucosal folds (Fig. 3*A* and 4*A*). Where two adjacent loops are involved, the abnormal gas shadows may be separated by a considerable width due to the hematoma (Fig. 2*B*). Whereas Nelson and Eggleston[4] found this sign in only 2 of 21 proven cases of bowel infarction, it was present in all 4 of our cases. One would therefore tend to consider it more specific for a hematoma than bowel infarction, especially if visualized in a patient receiving anticoagulants. It is conceivable that this important sign may be obliterated by complete obstruction. Its preservation is favored by the nonclotting blood which presumably dissects circumferentially in the submucosa or subserosa in a rather diffuse manner resulting in a uniform narrowing over long segments (Fig. 3*B*). Peristalsis is diminished or eliminated, tending to trap air in the involved bowel thereby facilitating its recognition. There is little localized mass effect as seen in traumatic hematomas where the blood can clot. If the hematoma occurs in a relatively closed space such as the retroperitoneal area, localized obstruction could conceivably occur by containment despite a lack of clotting.

CLINICAL FINDINGS

Abdominal pain, occurring in a patient known to be on anticoagulants should automatically lead to a search for intramural hematoma. This is especially true when there are visible signs of bleeding such as ecchymoses or hematuria. The clinical picture may vary from an acute abdominal emergency to nondescript cramping abdominal pain compatible with partial obstruction. Nausea, vomiting, distention and abdominal tenderness, either localized or diffuse, present in varying degree. Hematemesis and melena would not be unexpected. A drop in the hematocrit may occur out of proportion to the visible blood loss. An important diagnostic point in 3 of our cases was the rapid improvement in the abdominal pain and general condition of the patient after correction of the coagulation defect with vitamin K.

With the exception of Case III, none of our patients was in such straits as to require emergency surgery. In fact, when a diagnosis of mesenteric vascular occlusion was advanced in Case IV, the surgeon demurred since the patient was ambulatory and "just not sick enough for an occlusion." When the roentgen interpretation was considered with the clinical information, 2 of the 4 cases were spared surgery (Case II and IV), such was the reliance on the findings by the clinician. Though these 2 cases were not surgically verified, the roentgen appearance and subsequent clinical course were so characteristic that the diagnosis is inescapable.

ROENTGEN FINDINGS

Optimal demonstration of the extent of the pathologic process in intramural hematoma or in any of the diseases producing a "thick bowel" sign requires a barium study. Since many cases will present a suspicion of small bowel obstruction (Case II and III), either partial or complete, the use of barium may require some coaxing or re-education of referring physicians who are prejudiced

by surgical folklore relating to the use of barium in suspected intestinal obstruction. The use of water soluble contrast material may cause one to miss the diagnosis as in Case II.

Findings on routine abdominal roentgenograms vary from those of mechanical small bowel obstruction to a mild adynamic ileus. There was a suggestion in only 1 of the cases of a pseudotumor (Fig. 1*A*) such as one would expect in a closed loop obstruction due to volvulus or bands. Haziness due to intraperitoneal bleeding was found in 2 of the cases. The retroperitoneal soft tissues may be obscured by blood dissecting posteriorly in the mesentery (Fig. 1*A* and 4*B*). An unvarying finding was the presence of the narrow curving gas shadows described by Nelson and Eggleston[4] as a manifestation of thickened loops of bowel.

The classic findings on barium studies of an intramural mass and a crowding of the valvulae conniventes into a "coiled spring appearance"[1,2] were not seen in our cases. Our findings consist of a diffusely narrowed, relatively rigid segment of abnormal bowel, beginning and ending fairly abruptly. When outlined by barium, the narrowed lumen shows marked irregularity with sharp spicules of different lengths and widths formed by the barium entering the crevasses between hemorrhagic mucosal folds. The term "picket fence appearance" advanced by Wiot *et al.*[7] is a terse yet apt description. The fact the mucous membrane is preserved, though distorted, affords differentiation from a malignant process. The rapid return to normal eliminates the inflammatory entities noted earlier. The findings described above, when recognized, appear to be reasonably specific for intramural hematoma.

SUMMARY

1. Four cases of small bowel hematoma secondary to anticoagulant therapy are presented.

2. Three cases were diagnosed on routine abdominal roentgenograms from the "thick bowel" sign. This was actually present in all 4 cases.

3. This sign when recognized in a patient receiving anticoagulants is quite specific for intramural hematoma.

4. In 3 cases the process was reversible with medical management.

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SMALL BOWEL TRANSIT TIME OF BARIUM SULFATE PREPARATIONS AND IODINE CONTRAST MEDIA IN DOGS*

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BARIUM sulfate used in the examination of the gastrointestinal tract has undergone several modifications in recent years. These consist chiefly of micropulverizing the barium sulfate or of the addition of a suspending agent. Several clinical studies suggested a decreased transport time with these modifications in the barium sulfate.[1,4] We undertook a series of animal experiments to determine the effects of these changes in the barium sulfate preparation on the small bowel transit time. A water-soluble, iodine-containing contrast medium was included in the study because of its widespread use and reports of its accelerated passage through the gastrointestinal tract.[2,3]

PROCEDURE

A water-soluble, iodine-containing contrast medium (diatrizoate sodium 25 per cent) and three widely used commercial preparations of barium sulfate were used. One preparation was U.S.P. barium sulfate, a second had a suspending agent added, and a third contained only micropulverized barium sulfate. In a concentration recommended by the manufacturer, 100 ml. of each contrast material were placed through nasogastric tubes into the stomachs of 12 unanesthetized mongrel dogs. Each animal weighed between 11 and 39 pounds. Although the initially given preparation varied, each dog received all four preparations. These were given at intervals of several days after a 12 hour fast before each administration. Roentgenograms were taken at 30 minute intervals until the contrast medium reached the colon. No food or water was given to the animals during this time and they were unleashed in a large room between roentgenographic exposures.

TABLE I

TRANSIT TIME (IN MINUTES)

Dog	Diatrizoate Sodium 25 Per Cent	Micropulverized Barium Sulfate	Barium Sulfate with a Suspending Agent	U.S.P. Barium Sulfate
1	30	120	120	120
2	30	150	90	120
3	60	120	180	120
4	30	120	150	150
5	60	150	150	120
6	30	90	120	90
7	60	90	90	90
8	60	90	90	90
9	30	90	150	120
10	60	120	90	120
11	90	120	120	120
12	60	120	90	120

RESULTS

The time required for each contrast material to reach the cecum is given in Table 1. Gastric emptying of the three barium sulfate preparations was similar in all dogs, with only minor variations. No further observations or comparisons were made on the contrast materials.

SUMMARY AND CONCLUSIONS

The transport time through the small bowel in dogs of three barium sulfate preparations and a water-soluble, iodine-containing contrast medium was compared. The time required was consistently shorter with the iodine-containing contrast material. No significant difference was noted in the

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transit times of U.S.P. barium sulfate, micropulverized barium sulfate, and barium sulfate to which a suspending agent had been added.

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TUMORS OF THE TERMINAL ILEUM*

DIAGNOSIS BY RETROGRADE FILLING DURING BARIUM ENEMA STUDY

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TUMORS of the terminal ileum are rarely diagnosed preoperatively unless advanced disease is present. In most instances obstruction has supervened and a correct diagnosis is possible only at surgery.

Since a significant percentage of such tumors are malignant or potentially malignant, *e.g.*, carcinoid, lymphoma, sarcoma,

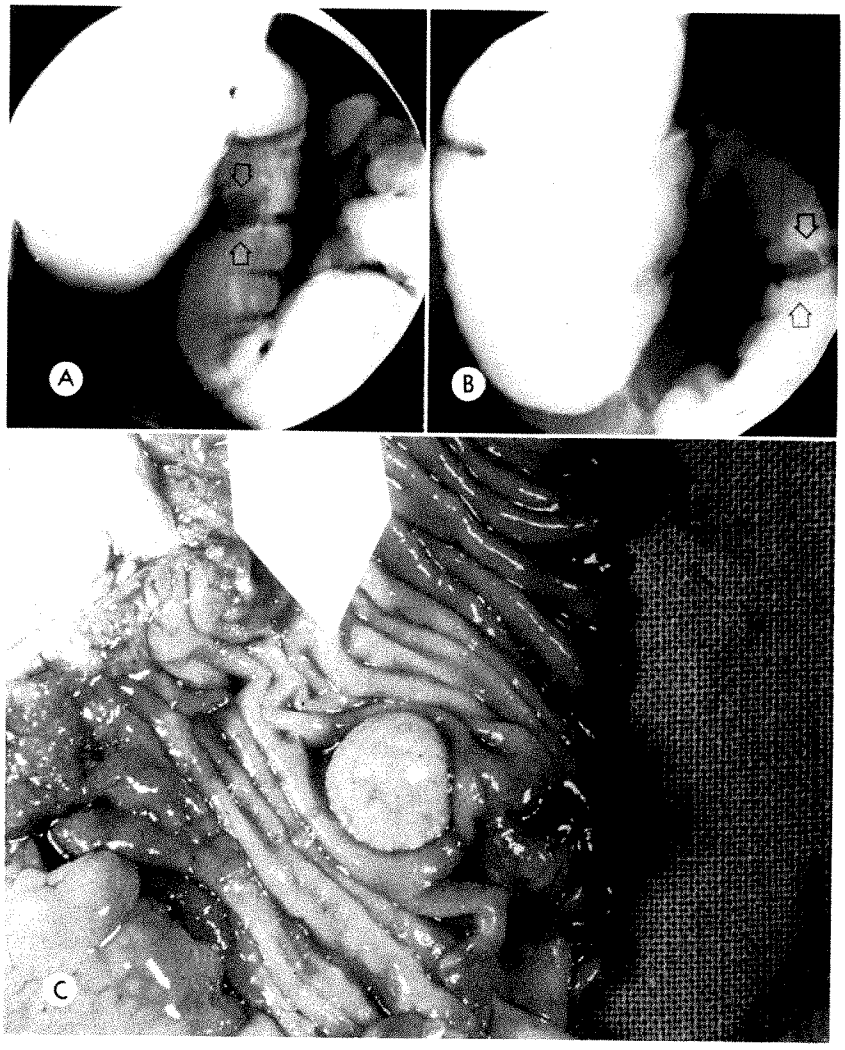


FIG. 1. Forty year old white female with carcinoid of the terminal ileum with metastases to the ileocolic lymph nodes and blood vessels. The patient's only complaint was recurrent peri-umbilical pain. (*A*) Roentgenogram showing the 1.5 cm polypoid tumor in the terminal ileum. (*B*) Identical findings are demonstrated on a second examination. (*C*) Photograph of the surgical specimen.

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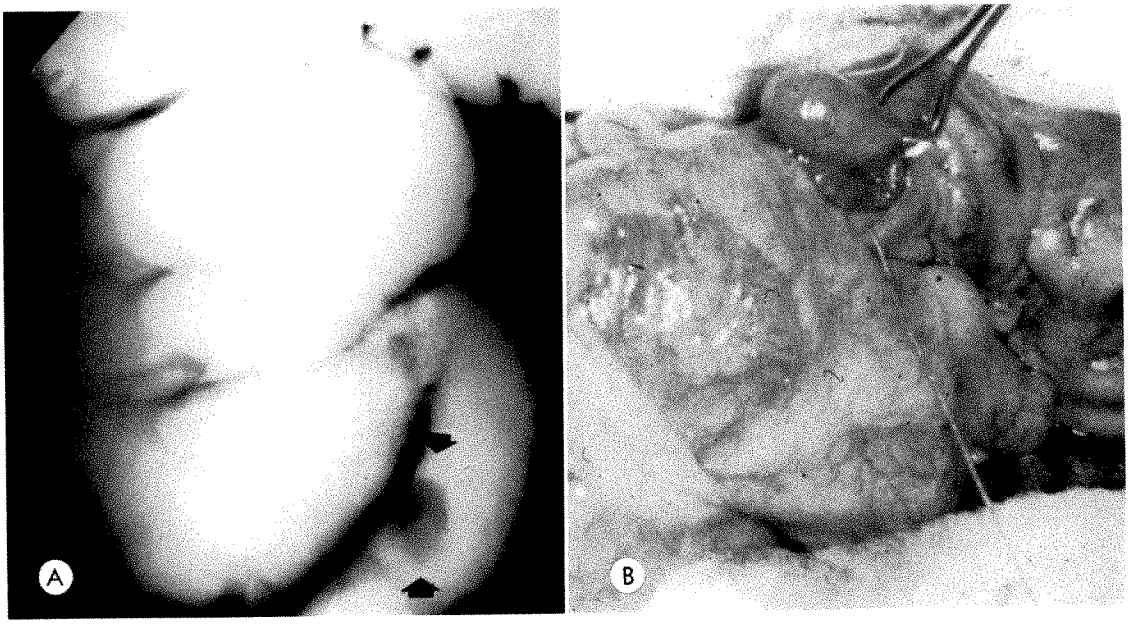


Fig. 2. Thirty-eight year old white male with a pedunculated lipoma of the terminal ileum. The patient had vague, nonspecific symptoms. (*A*) High kv. spot compression roentgenogram demonstrating the polypoid tumor in the terminal ileum. The same findings were noted on a second examination. (*B*) Photograph of the surgical specimen.

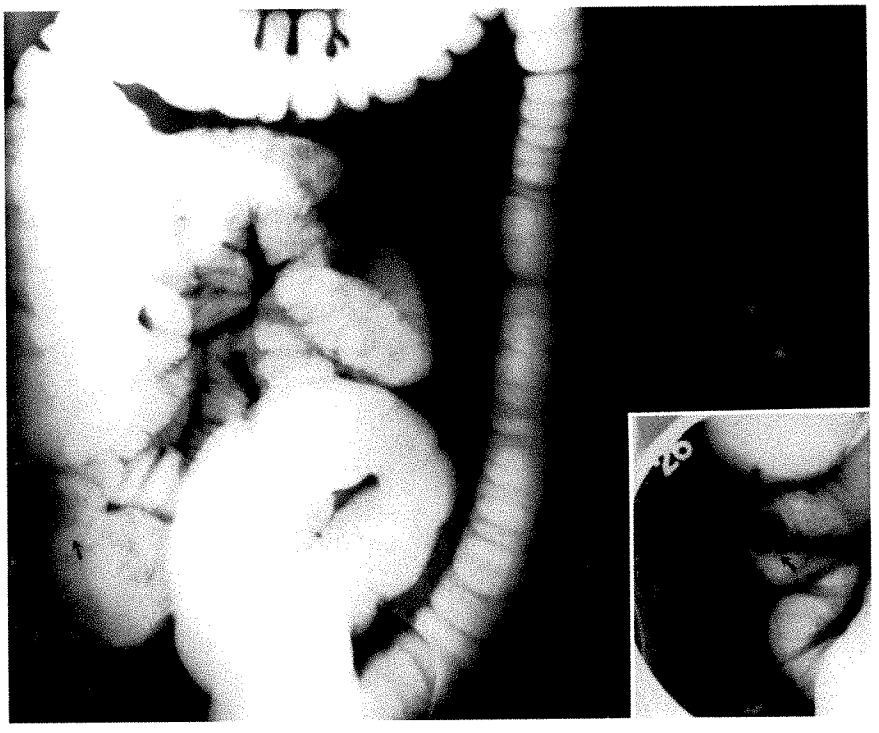


Fig. 3. Forty year old white male with a 2 cm. tumor of unknown etiology in the terminal ileum. The patient's symptoms were nonspecific. The tumor was demonstrated on two separate examinations. Patient refused surgery.

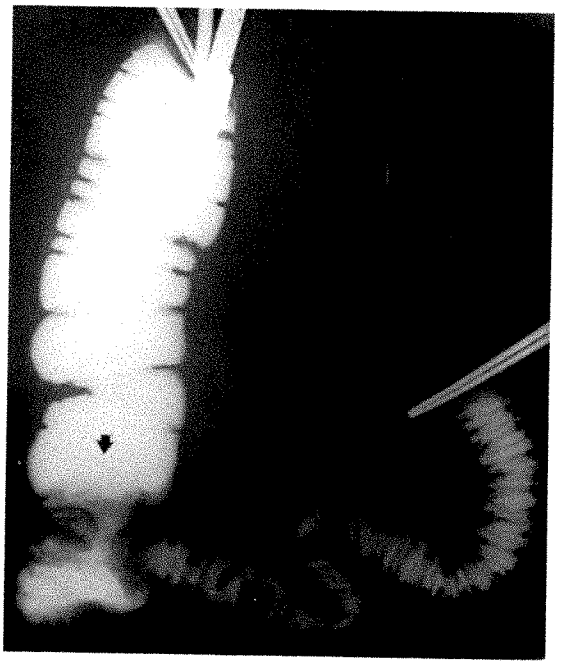


Fig. 4. Sixty year old white female who had an annular carcinoma of the cecum. Roentgenogram of the surgical specimen shows the 1 cm. pedunculated adenoma.

and, since tumors of this region predispose to acute obstruction and strangulation as a result of intussusception, an early diagnosis is important.

METHOD

Retrograde filling of the terminal ileum during barium enema study permits an exact evaluation of this segment of the gastrointestinal tract. The absence or presence of various disease states, inflammatory or neoplastic, can be determined easily and accurately if there has been good preparation for the procedure and filling of the ileum is possible. The utilization of high kilovoltage or overpenetration techniques combined with large field spot compression studies allows an amazing visualization of anatomic and pathologic detail.

MATERIAL

Roentgenograms of 4 cases, 3 in which small polypoid tumors were discovered in the terminal ileum and 1 in which a small tumor was diagnosed by roentgenographic examination of the surgical specimen prior to examination by the pathologist, are presented (Fig. 1 through 4, inclusive). In the last case an annular carcinoma was discovered in the cecum during a barium enema study and a right hemicolectomy was performed. Retrograde filling of the ileum was not accomplished in this case.

SUMMARY

The terminal ileum is a favorite site of various disease states, inflammatory and neoplastic. These processes are diagnosable roentgenographically if retrograde filling of the terminal ileum is obtained during the barium enema examination. Roentgenograms are presented showing the effectiveness of large field, high kv. spot studies in the detection of small tumors of the terminal ileum.

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ROENTGEN DEMONSTRATION OF GANGRENE OF THE STOMACH AND INTESTINE*

A LATE FINDING IN INFARCTION OF THE GASTROINTESTINAL TRACT

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A CASE is presented which shows an unusual linear and intramural collection of gas in the stomach and intestine. There was an associated distention of the gut which contained large amounts of fluid. This combination of findings is probably pathognomonic of gangrene of the gut in a late stage. There was also an unusual distribution of gangrene with involvement of the stomach as well as the entire small bowel and colon down to a recent anastomosis of the descending colon and sigmoid. This atypical pattern provided an interesting opportunity for review of the subject of intra-abdominal vascular occlusion.

REVIEW OF ANATOMY

According to the classic description, the celiac artery arises from the front of the aorta immediately below the diaphragm. It is a large artery, only one-half inch in length, and divides into branches which supply the liver, stomach and spleen. The superior mesenteric artery arises from the aorta about one-half inch below the celiac artery and opposite the first lumbar segment. Its course is obliquely downward and forward, making an acute angulation as it passes anterior to the left renal vein. The superior mesenteric artery supplies part of the duodenum and pancreas, the entire small bowel distal to the duodenum, and the large intestine nearly as far as the splenic flexure.

There are many and frequent variations in the origin, course, distribution and anastomoses of the intestinal vessels.[15] A rather contiguous origin of the superior mesenteric artery and the celiac artery is frequent, but a common trunk is rare. The hepatic artery, or an accessory hepatic artery, originates from the superior mesenteric artery in 17 per cent of the cases. The gastric and splenic arteries have less frequent variation in origin.

There is some anastomosis between the celiac artery branches and the superior mesenteric artery branch via the gastroduodenal artery and the pancreaticoduodenal arteries. Occlusion of the superior mesenteric artery with resultant gangrene usually begins in the proximal jejunum rather than in the distal duodenum which is the most proximal part of its supply area.

A variable number of intestinal arteries take origin from the superior mesenteric artery. Each artery courses fairly straight between the layers of the mesentery and then unites with adjacent branches to form an arcade. The arteriae rectae, small straight vessels, arise from the terminal arcades and supply the intestinal wall. The arteriae rectae are usually end arteries, but occasionally form definite intermesenteric anastomoses. Some segments of small intestine are vascularized by arteriae rectae coming from two terminal arcades: one arcade supplying the gut anteriorly and the other arcade sending branches to the posterior surface. Within the bowel wall the arteriae rectae communicate with one another and form a continuous network of arterial anastomoses.

The inferior mesenteric artery arises from the front of the aorta about one-half inch above the bifurcation of the aorta and supplies the colon distal to the splenic

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flexure. In the absence of the middle colic artery, the inferior mesenteric artery is the major blood supply of the transverse colon and may supply it to the hepatic flexure.

The marginal artery of Drummond links the arcades of the right and middle colic artery (branches of the superior mesenteric artery) with the left colic artery (a branch of the inferior mesenteric artery). The anastomosis is sufficient to supply that segment of colon deprived of its usual vascular source by ligation of any one of the colic arteries or the inferior mesenteric artery. The inferior mesenteric artery is commonly ligated during a cancer operation of the distal colon.

There is a potential third blood supply to the colon by way of the anastomosis between the superior rectal (hemorrhoidal) artery, which originates from the inferior mesenteric artery, and the middle and inferior rectal (hemorrhoidal) arteries, which ultimately are supplied by the internal iliac arteries.

The venous drainage of the intestine and colon closely parallels the arterial supply in number, origin, distribution and variation. The superior mesenteric vein and splenic vein unite to form the portal vein. The inferior mesenteric vein enters the portal system by draining into the splenic vein, the superior mesenteric vein or the origin of the portal vein.

The superior rectal (hemorrhoidal) vein flows into the inferior mesenteric vein and then drains into the portal system. The middle and inferior rectal (hemorrhoidal) veins drain into the inferior vena cava via the internal iliac veins.

In portal hypertension the flow may be reversed so that portal blood may pass into the inferior mesenteric vein and into the rectal plexuses, where it is carried away to the inferior vena cava through the inferior rectal veins and the internal iliac veins.

MECHANISM

Gangrene results from vascular occlusion, whether arterial, venous or a combination of the two. Gangrene of the gut is a local phenomenon in approximately 65 per cent of the cases and usually is caused by local vascular occlusion resulting from the following mechanical situations: external hernia, adhesions, volvulus, intussusception, or infection which causes swelling, and impaired circulation as in appendicitis.

Vascular occlusion is the primary factor in 35 per cent of the cases of gangrene of the gut, and it is the most common single etiologic factor.[19] Primary vascular obstruction has been more specifically designated as mesenteric vascular occlusion. There are several varieties of vascular occlusion.

1. Completely reversible vascular occlusion of short segments of the colon has been reported by Schwartz *et al.*[17] The process is a disease of the small vessels and may be either a primary vascular disorder or secondary to another pathologic process. The pathologic changes are due to submucosal hemorrhage and pericolic fat inflammation. There is a complete resolution after a few days. No late sequelae are found. A similar case of localized infarction of the colon was previously described by Engelhardt and Jacobson.[6]

2. Wolf and Marshak[20] reported short segmental infarction of the small bowel without gangrene, but with late stenosis of the involved area. Vascular occlusion of small vessels in their cases resulted in severe injury to the more sensitive mucosa, but there was viability in the less sensitive muscular fibers and the connective tissue elements of the bowel wall. Viability of a portion of the gut presumably was retained by anastomoses in a relatively short involved segment of bowel. Late changes in the intestine were atrophy of mucosa, fibrosis of the bowel wall and absence of the valvulae.

3. Recurrent vascular occlusion of the small bowel was described by Cregg and Smith.[2] A second episode of mesenteric infarction developed 9 days following a segmental resection of the bowel for hemorrhagic infarction. The second area of gangrenous bowel was 30 cm. proximal to the original resection. The patient had a

history of arteriosclerotic disease and auricular fibrillation for several years. It is probable that embolic phenomenon was the underlying factor rather than extension of the original thrombus since the second infarcted area was separated from the previous resection by normal bowel.

4. By far the most common type begins or progresses to complete vascular occlusion producing gangrene. The superior mesenteric system is involved in over 90 per cent of the cases. The obstruction usually involves the vessel near its origin. Occlusion of the arterial system is slightly more common than of the venous structures. In about 25 per cent of the cases, it is impossible to localize the source of primary obstruction to one side of the circulation system since the process is frequently progressive and involves both arterial and venous segments in the advanced stages which are usually observed at autopsy.

Arteriosclerosis is a potent contributing condition in some cases. Narrowing of the superior mesenteric artery results in slowed circulation and makes the artery vulnerable for occlusion from any further vascular insult. Anatomic studies show that arteriosclerosis in the superior mesenteric artery usually occurs 5 to 15 mm. from the origin of the artery at the site of acute angulation.[3,5] Occlusion of the inferior mesenteric artery is usually secondary to an arteriosclerotic plaque in the wall adjacent to the ostium. Occlusion of the vessel seldom produces infarction of the colon unless there is associated vascular disease.

Embolism originating from the left side of the heart is probably the most common cause of mesenteric infarction. The embolus may come from an area of myocardial infarction, rheumatic valvular heart disease or bacterial endocarditis.[4,8] A detached atheromatous plaque from the aorta is sometimes the source of an embolus.

Portal vein obstruction and hypertension of this system is often a causal factor in mesenteric venous thrombosis. Therefore portal cirrhosis is frequently complicated by thrombosis of the portal vein and hepatic cirrhosis is present in a majority of the patients with portal thrombosis. Schistosomiasis is an important factor in certain areas of the world.

Less common conditions causing vascular occlusion of mesenteric vessels are: periarteritis nodosa, migrating thrombophlebitis and intraperitoneal sepsis. The latter usually produces venous thrombosis and was a common cause prior to the advent of antibiotics. Rare causes are thromboangiitis obliterans, syphilis, polycythemia vera, trauma and alcoholism.

The postoperative and especially the postsplenectomy state shows an increased incidence of mesenteric vascular occlusion. The mechanism following surgery is often obscure.

Consideration of the possible etiologies is important in leading one to suspect mesenteric vascular occlusion, especially in the gradually developing insidious case.

The bowel wall is thickened and engorged with blood following occlusion of a major mesenteric vessel. The adjacent mesentery is swollen and contains areas of hemorrhage and distended veins. The intestinal mucosa becomes necrotic and an inflammatory exudate appears on the serosa. There is thick dark blood in the lumen and bloody fluid in the peritoneal cavity. The bowel is dark in color. Gangrene and peritonitis develop later.

CLINICAL FEATURES

The onset of mesenteric thombosis is often acute with abdominal distention, severe pain and marked vomiting. Discomfort is characteristically disproportionately severe for the clinical findings. Shock accompanies an acute onset and varies with the extent of the occlusion. Diarrhea is common and sometimes the stool contains small amounts of blood; constipation has occurred in some cases. Peristalsis is usually diminished. Fever and elevation of the white blood cell count occur later in the disorder.

Other cases of mesenteric thrombosis develop slowly over a period of a week or more and with vague symptoms of abdominal distress.

Mesenteric infarction is most commonly confused with acute pancreatitis, perforated peptic ulcer and acute cholecystitis.

Sudden abdominal pain in a patient with serious heart disease, severe arteriosclerosis or a history of recent abdominal surgery is suggestive of mesenteric infarction. Aspiration of bloody ascitic fluid strengthens the probability of vascular occlusion.

Early diagnosis and prompt surgery offer the only hope of recovery after gangrene has developed. Resection of a large segment of gangrenous bowel has been successful. Embolectomy and possible bypass arterial surgery may be successful if done early enough.[9,13] The operative mortality is high; the mortality rate with expectant treatment is almost 100 per cent.

ROENTGEN FINDINGS

A roentgenogram of the abdomen frequently demonstrates nonspecific distention of the small bowel; however, about one-third of the cases of mesenteric infarction do not have any abnormal findings. The segment of distended intestine does not correspond closely with the area of infarction.

Certain patterns and distributions of gas in the bowel suggest the diagnosis of mesenteric infarction.

Segmental reversible vascular occlusion of the colon produces "thumb printing" of the colon wall due to nodulation with spasm.[17] "Pseudotumor" deformity results from nodulation without spasm and is caused by tumefactions of submucosal hemorrhage. The bowel returns to a normal state after a few days.

In the small bowel there may be late changes from segmental occlusion. There is tubular narrowing of a segment of intestine from 3 to 6 inches in length. The mucosal pattern is effaced. There is a fairly abrupt but smooth change from the constricted segment to the normal bowel.

The more usual cases of mesenteric infarction have been extensively described. The cases reported by Harrington and Rendich[10,16] showed dilatation of the small bowel and colon proximal to the splenic flexure without mechanical obstruction of the splenic flexure on barium enema studies. This was considered as a sign-complex suggesting superior mesenteric vascular occlusion; it has been an uncommon finding in other series of cases.

Frimann-Dahl[7] described cases of mesenteric occlusion with distended small bowel containing large amounts of gas and fluid which simulated mechanical obstruction. Distention progressed rapidly and aided in differentiating vascular occlusion from mechanical obstruction. Barium given by mouth showed thickening and rigidity of the mucous membrane from edema. Transit time of the barium meal was prolonged.

Thickening of the bowel wall with an accompanying narrow lumen outlined by air was described by Cregg and Smith[2] in a case of recurrent infarction of the ileum. The wall of the intestine was 1 cm. thick due to edema and hemorrhage of the bowel. Edematous mesentery produced the appearance of a soft tissue mass on roentgenograms.

Wang and Reeves[18] and Nelson and Eggleston[14] reported the roentgen findings in cases of mesenteric vascular occlusion. In both series there were a few cases of marked thickening of the bowel wall with a narrow intraluminal air shadow. The gas shadow was straight or slightly curvilinear and remained constant with a change in position of the patient. Sometimes there were parallel gas-filled loops like a "cluster of bananas." Subacute or chronic cases developed narrowing and fixation of the bowel wall simulating regional enteritis or ulcerative colitis.

No report could be found of gas in the stomach wall due to vascular occlusion. Gas in the stomach wall is rare. It was described

by Lumsden[12] in the following conditions: emphysematous gastritis, cystic pneumatosis and interstitial gastric emphysema. Emphysematous gastritis is a special variety of severe phlegmonous gastritis. Cystic pneumatosis is caused by benign gas filled cysts on the outer wall of the alimentary tract. These cysts are usually in the small bowel and rarely in the stomach. Interstitial gastric emphysema may follow gastrostomy and does not produce clinical symptoms. Cancelmo[1] reported a case of intraluminal gas in the stomach following a feeding jejunostomy for inoperable carcinoma of the lower esophagus. It was postulated that postoperative vomiting forced air through an incomplete perforation of the esophagus and that the gas passed into the gastric wall.

A bizarre case of spontaneous passage of over one-half of the colon following mesenteric thrombosis was reported by Lister and Jungmann.[11] Fragments of the gangrenous colon passed through a fecal fistula and through the rectum.



FIG. 1. An arrow points to a large malignant tumor in a loop of sigmoid colon. A second arrow indicates a benign polyp on a stalk in the distal descending colon.

REPORT OF A CASE

M. D., a 63 year old white female, was admitted to the hospital with symptoms referable to the colon. A barium enema study on July 1, 1960 revealed marked diverticulosis of the sigmoid and probable neoplasm in the mid-sigmoid. Recheck roentgenographic studies confirmed a tumor 4 cm. in diameter in the mid-sigmoid and demonstrated a polyp 2 cm. in size in the distal descending colon (Fig. 1). She had had a hemorrhoidectomy, sympathectomy and cholecystectomy prior to 1954. She was on medication for severe hypertensive heart disease. Her blood pressure on admission was 210/120.

A sigmoid resection with end-to-end anastomosis, total colonoscopy and polypectomy were done on July 15, 1960. Her blood pressure during operation ranged from 110–180/70-120. The pathology report was: (1) papillary adenoma of the sigmoid with development of mucoid adenocarcinoma; (2) metastatic mucoid adenocarcinoma of mesocolic lymph nodes; and (3) pedunculated adenoma of the colon. On the fifth postoperative day, she passed a large quantity of stool and apparently was convalescing satisfactorily. On the sixth and seventh postoperative days, she gradually developed progressive abdominal distention and low grade spiking fever. There was minimal tenderness and pain in the lower abdomen. On the evening of the seventh day, she had diarrhea and was very nauseated. There was rather marked distention of the abdomen and she felt very ill. The white blood cell count rose from 12,000 on July 18, 1960 to 35,400 on July 23, 1960.

Roentgenographic examination of the abdomen on July 23, 1960 showed severe distention of multiple fluid containing loops of small bowel and a most unusual band of air 2 mm. wide in the stomach and small bowel wall (Fig. 2 and 3). The air shadow was continuous and uniform, and was unaltered by changes in body position.

Abdominal exploration on the same day revealed gangrene of the entire small intestine. There was free blood-tinged peritoneal fluid. The previous sigmoid anastomosis was intact. A loop of small bowel was exteriorized and vented by a rectal tube. Brownish and blood-

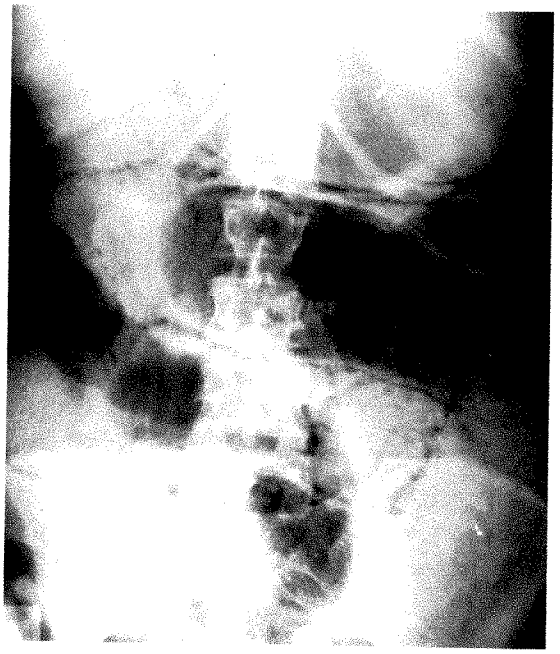


Fig. 2. A supine roentgenogram of the abdomen shows a uniform, continuous, narrow zone of intramural air in the wall of the stomach and proximal intestine. The small bowel is severely dilated and atonic.

tinged fluid exuded from the lumen of the small bowel. A presumptive diagnosis of thrombosis of the superior mesenteric artery was made. She expired 2 days later.

Autopsy revealed marked infarction and necrosis of the stomach, small intestine and colon. These organs showed severe black discoloration. The colon on the proximal side of the anastomosis in the rectosigmoid area was completely necrotic, while the colon wall distal to the anastomosis was viable. There were large areas of infarction in the liver, spleen and pancreas. The esophagus was negative. There was severe atherosclerosis of the aorta with beginning ulceration in several foci of calcification. The celiac axis was relatively small. A rather recent thrombus occluded the lumen and extended into the vessel about 3 cm. The superior mesenteric artery was very small. Atherosclerotic changes were found proximally in this vessel. The inferior mesenteric artery had been ligated during surgery on July 15, 1960. Microscopic studies confirmed the gross findings. Other findings at autopsy were: marked parenchymatous degeneration of the adrenal glands, fatty infiltration of the right ventricle of the heart, small areas of myocardial fibrosis, and acute peritonitis.

COMMENT

The combination of intramural gas, marked distention of small bowel and a large amount of fluid in the small bowel is probably a sign-complex that is specific for extensive vascular occlusion in the late stage.

The case reported is of particular interest in that there was infarction of the gut from the proximal stomach to the recent anastomosis of the descending colon to the rectum. This distribution is explained by the findings at autopsy of developmentally small celiac and superior mesenteric arteries, further narrowing from atherosclerosis, and thrombosis of the celiac artery. Circulation to the superior mesenteric artery became inadequate following ligation of the inferior mesenteric artery and elimination of the collateral flow from the superior rectal arteries following resection of the sigmoid. Slowed circulation from a drop in



Fig. 3. There are multiple fluid levels on an erect roentgenogram of the abdomen. There is no change in the pattern or distribution of intramural air with varied positions of the body.

blood pressure in a severely hypertensive patient and vasospasm secondary to thrombosis of the celiac artery were additional factors in producing infarction of the small bowel, even though the superior mesenteric artery was not occluded.

The colon below the sigmoid anastomosis was adequately supplied from the superior rectal arteries and was not infarcted.

Intramural gas was probably produced by gas forming bacteria in the gastrointestinal tract migrating into devitalized stomach and intestinal wall.

SUMMARY

1. A case of massive infarction of the gastrointestinal tract following surgery of the colon is reported. The celiac axis was thrombosed. The superior mesenteric artery was abnormal and diseased, but was not occluded.

2. A narrow zone of intramural gas in the stomach and intestine is a rare, late finding in gangrene of the gastrointestinal tract.

3. The sign-complex of intramural air in the small bowel and stomach, marked dilatation of the small bowel and a large amount of fluid within the intestinal tract is suggestive of extensive vascular occlusion of the gastrointestinal tract.

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FIBROSIS AND STENOSIS OF THE DESCENDING COLON AND SIGMOID FOLLOWING OCCLUSION OF THE INFERIOR MESENTERIC ARTERY*

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STENOSIS of the colon rarely occurs following occlusion of the inferior mesenteric artery because the abundant collateral circulation prevents infarction and ischemic necrosis of the bowel. However, the marginal artery of Drummond, which serves as an anastomosis between the middle colic branch of the superior mesenteric artery and the left colic branch of the inferior mesenteric artery, may be congenitally absent. Then the collateral blood supply may prove inadequate to prevent infarction of the colon. However, this infarction is usually a catastrophic event; the mortality rate is very high and few patients survive, without resection, to develop fibrosis and stenosis of the colon.

In the case reported it is our purpose to describe and emphasize the roentgen findings following occlusion of the inferior mesenteric artery and infarction of the descending colon and sigmoid. Although there have been several descriptions of the early roentgen changes following infarction of the left colon[3,7] and a few reports of segmental stenotic lesions of the rectum or sigmoid[6,9,10,14,16] which developed subsequent to infarction, we have found no presentation of stenosis of the major portion of the descending colon and sigmoid as occurred in our patient.

REPORT OF A CASE

On April 4, 1962 a well nourished, intelligent, 62 year old business man was admitted to Evangelical Deaconess Hospital complaining of severe lower abdominal pain. During that afternoon the patient felt fine and had worked several hours in his garden. Abdominal pain came on suddenly, shortly after his evening meal; it was more severe on urination, but there was no blood in the urine.

Physical Findings. Initially the pertinent findings were limited to the abdomen which was tender but not rigid, and no masses were palpable. The patient was conscious, coherent and afebrile. His blood pressure was 170/100 and his pulse was 102.

Laboratory Findings. On admission the hematocrit was 47 per cent, hemoglobin 15.2 gm., and the white blood cell count was 21,000. Other tests, including serum sodium, potassium chlorides, alkaline phosphatase, and CO_2 combining power, blood sugar and blood urea nitrogen were within normal range. The cephalin flocculation test was 4+ in 48 hours. Urinalysis was normal except for the presence of an occasional red blood cell. The electrocardiogram also was normal.

Course in Hospital. Because the patient complained of inability to urinate, a Foley catheter was inserted soon after admission. The following morning abdominal pain increased and his abdomen was more distended. A surgical consultant noted a mass in the left lower quadrant —seemingly adherent to the sigmoid colon. The clinical impression was that a sigmoid diverticulum had perforated and a pericolic abscess was developing. A Levine tube was inserted and supportive therapy, including distrycillin and intravenous terramycin, was started.

Roentgenograms of the abdomen (Fig. 1) taken the morning following admission with the patient recumbent and in the left decubitus position revealed a nonspecific gas pattern with distended loops of small intestine. The descending colon contained gas, had smooth margins and some normal-appearing haustral markings were demonstrated. The appearance seemed consistent with an adynamic ileus of unknown cause.

On the second hospital day a rectal examination revealed a dark, sanguineous, semi-solid

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fluid on the examining finger, and areas of ecchymosis were noted about the anus. Immediately prior to this examination, the patient had received a soap suds enema with return of some "dark bloody water" containing small flecks of brown stool. Sigmoidoscopy was then performed. The instrument was passed with ease to 20 cm. without evidence of obstruction. But very significant changes were noticed throughout the examined segment. The mucosa was pale gray and non-glistening with scattered, irregular, dark areas due to submucous ecchymoses. The appearance suggested local trauma possibly secondary to the enema. No brown stool or blood was seen. Attempts to advance the sigmoidoscope beyond 20 cm. were not successful because the patient became restless and uncooperative. The examination was discontinued without obtaining a biopsy.

For the next 2 days abdominal distention continued. The patient passed large amounts of foul-smelling flatus and had numerous liquid, brown stools. On April 9 roentgenograms of the abdomen (Fig. 2) showed an increase in small bowel distention with scattered collections of gas

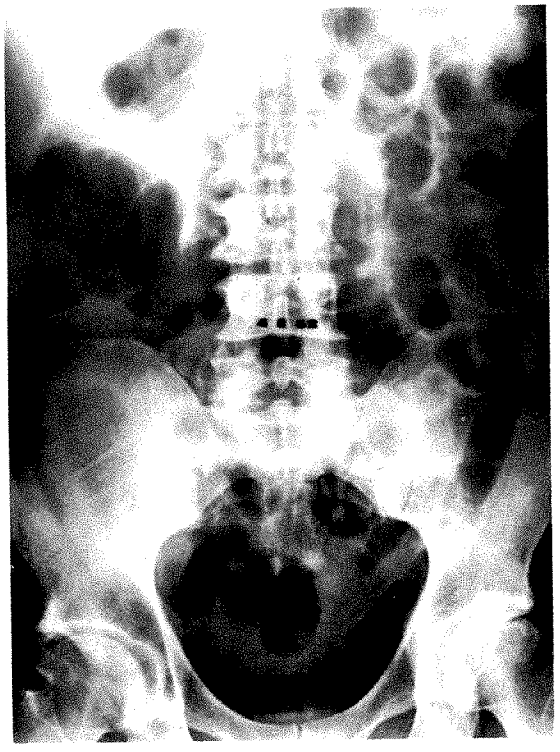


Fig. 1. Recumbent roentgenogram of abdomen on April 5, 1962. Nonspecific gas pattern suggests adynamic ileus.

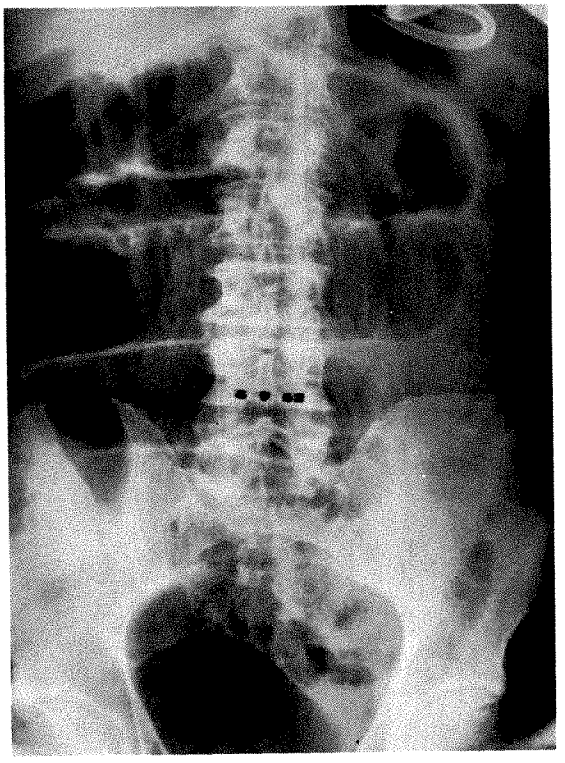


Fig. 2. Recumbent roentgenogram of abdomen on April 9, 1962. Numerous dilated loops of small intestine in step-ladder pattern. Small amount of gas in descending colon. Note gas in urinary bladder.

in the descending colon and sigmoid. In addition, the bladder appeared to be distended with gas. The following morning a small amount of contrast medium was introduced into the bladder and an upright roentgenogram was made (Fig. 3). A gas-fluid level was noted between the gas and urine, and, in addition, a fluid-fluid level was present at the interface between the urine and contrast medium. Since it was assumed clinically that a sigmoid diverticulum had ruptured with subsequent inflammatory perforation of the bladder, it was considered advisable to examine the sigmoid and descending colon by barium enema, using maximum caution to avoid additional trauma to the colon. The study (Fig. 4) disclosed no diverticula and no site of perforation was found. The walls of the upper rectum and sigmoid were slightly irregular and the haustral markings were wide and coarse. The mucosal pattern was effaced. Along the sigmoid, where a mass was palpable, the colon was very irritable and did not retain barium. No ulcerations were noted along the

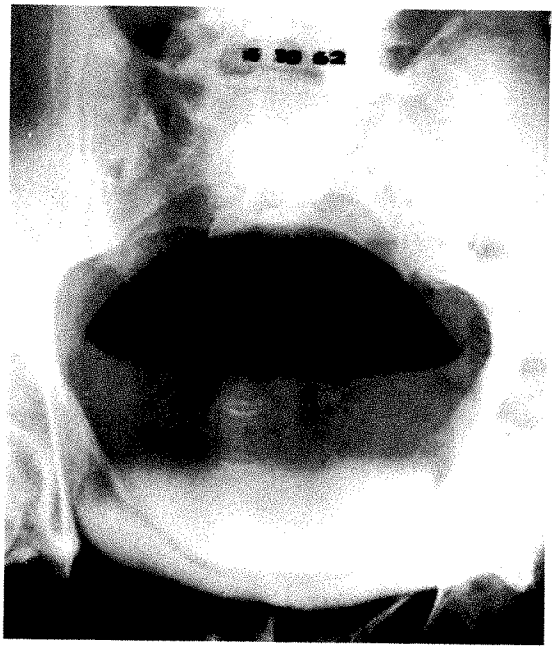


Fig. 3. Erect roentgenogram of the bladder made on April 10, 1962, after injection of contrast medium.

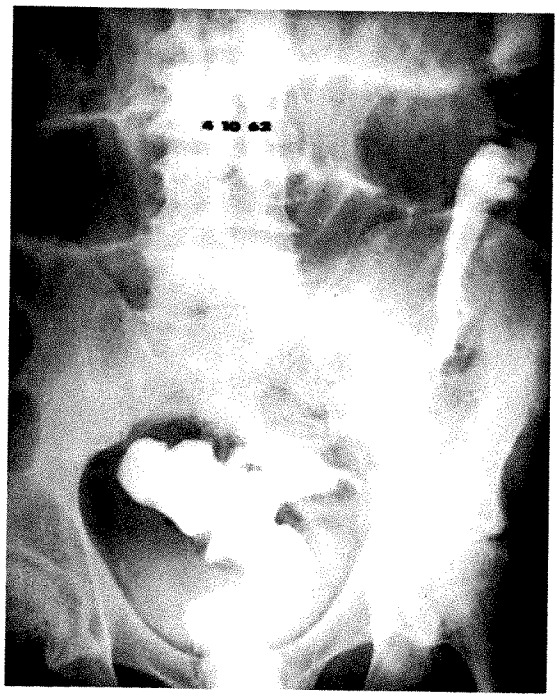


Fig. 4. Barium study of rectum, sigmoid, and descending colon on April 10, 1962. Walls slightly irregular, mucosal markings effaced and some wide and irregular haustra present. Proximal sigmoid irritable and did not retain barium in the region of the palpable mass.

margins of the colon and no pseudopolyps[1,7] were recognized. No effort was made to fill the entire colon with barium due to fear of perforation. The significance of the roentgen changes in the colon was not appreciated at the time, and specific diagnosis was not suggested.

For the next 10 days his condition improved slowly. As earlier, he passed large amounts of flatus and continued to have many liquid, brown stools. While the patient was walking about on April 20, he was aware of strands of tissue protruding from his rectum. These were removed and the pathologist described them as "Necrotic mucosa and submucosa of the rectum or colon." For the next few days more strands appeared. On April 24 roentgenograms of the abdomen (Fig. 5) revealed large numbers of tiny gas bubbles throughout the descending colon and proximal rectum—suggesting pneumatosis intestinalis. Finally, on April 26 the patient passed a large soft stool encased in a long mucosal cast of the descending colon and sigmoid.

On May 5, a month following admission, a barium enema examination was done. The strik-

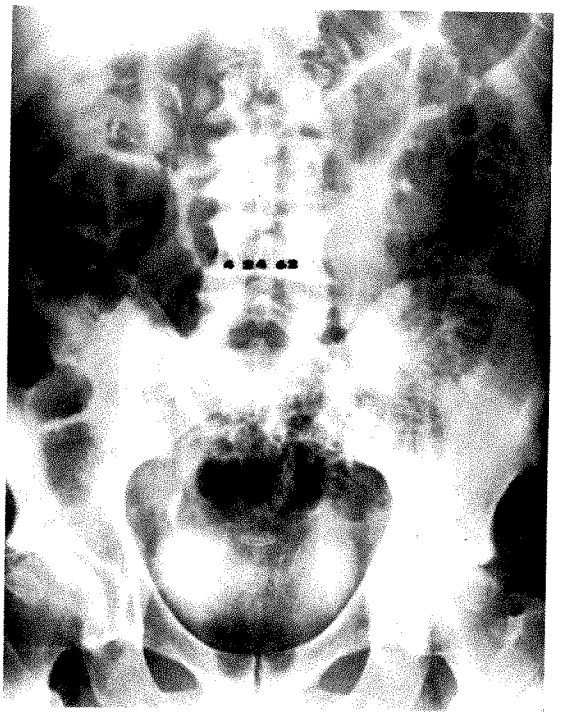


Fig. 5. Intravenous pyelogram on April 24, 1962. No urinary tract abnormality. Small bubbles of gas can be seen throughout descending colon, sigmoid and proximal rectum.

ing changes (Fig. 6) included: narrowing of the descending colon and sigmoid; loss of haustral markings; complete absence of mucosal markings in the involved segment; and sharp demarcation between the abnormal and normal bowel with cone-shaped transition to the normal proximal bowel. For the first time, the roentgenologists were convinced that the changes in the colon were due to infarction following occlusion of the inferior mesenteric artery.

Although the patient had lost 27 pounds, he was improving slowly. It was considered advisable to discharge him on May 5, 1962 to allow time for collateral circulation to develop before the abnormal bowel was resected.

While still an out-patient, he returned for a progress barium enema examination on June 26, almost 3 months from the onset of illness. At this time (Fig. 7) the descending colon and sigmoid showed marked stenosis, the lumen measuring 7 mm. In the interval since the last examination, considerable shortening of the colon had occurred causing the splenic flexure to be lower than it was on the earlier study.

Second Admission. On August 13, 1962 the patient entered the hospital again and the barium enema examination was repeated the next day (Fig. 8). The stenosis and shortening had increased since the examination of May 5, 1962.

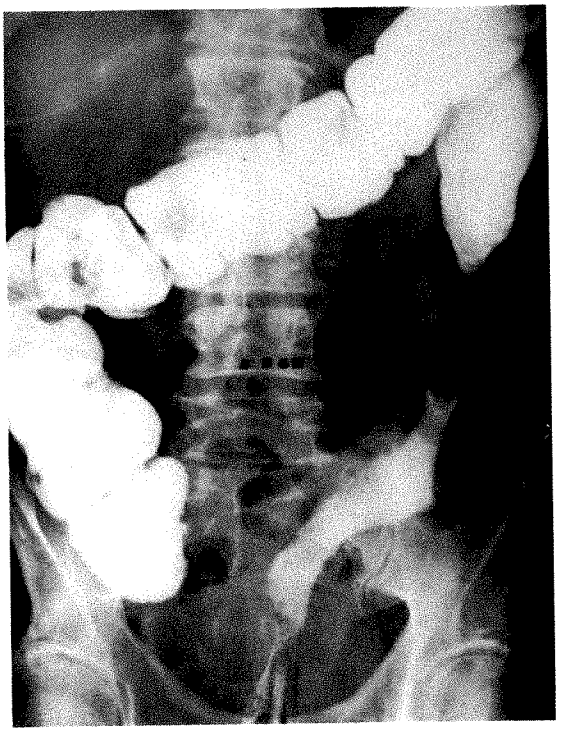


Fig. 6. Barium enema study on May 5, 1962. Descending colon and sigmoid smooth and narrow and the mucosa is destroyed. Cone-shaped demarcation between normal and abnormal bowel.

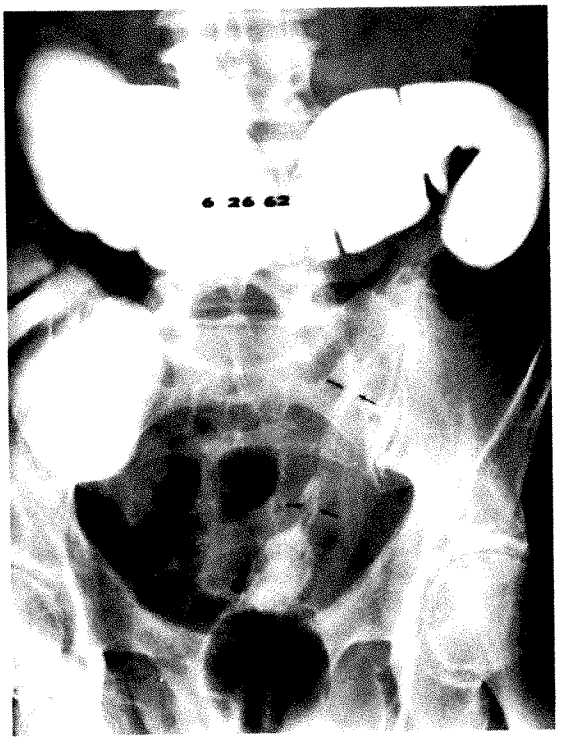


Fig. 7. Barium enema study on June 26, 1962. Marked stenosis of descending colon and sigmoid. Mucosa completely destroyed. Sharp demarcation between normal and abnormal bowel. Note splenic flexure now located just above iliac crest.

While convalescing at home he had continued to lose weight—a total of 45 pounds—and diarrhea had continued. On August 17 the descending colon and sigmoid were resected and an anastomosis was established between the transverse colon and rectum. On incising the peritoneal reflections of the colon, the surgeon encountered pus. He also observed a moderate-sized aneurysm of the abdominal aorta extending from a point 2 or 3 cm. distal to the renal arteries to the bifurcation of the aorta.

The excised colon segment measured about 10 cm. in length and was described as brown, discolored cord-like tissue. The summary of the pathologic report of the resected colon was as follows: "Chronic ulcerative, fibro-stenotic lesion of the descending colon and sigmoid, probably secondary to thrombosis or thromboembolism and organization of medium caliber

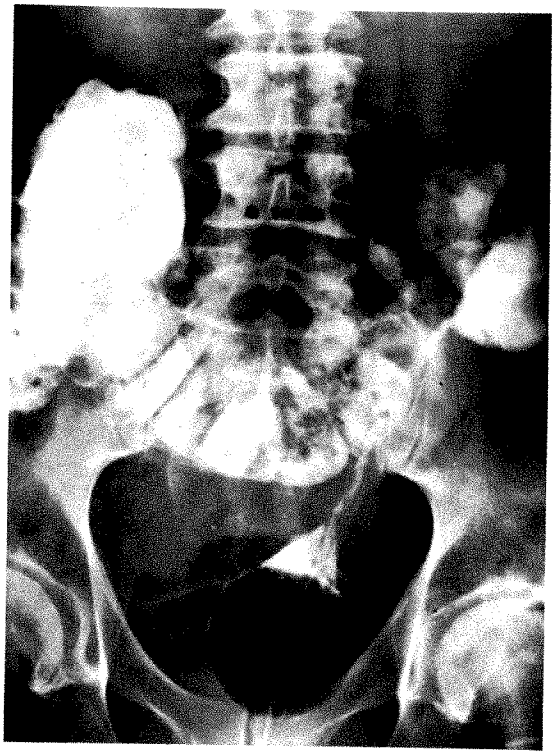


Fig. 8. Barium enema study on August 14, 1962. Marked stenosis and shortening of descending colon and sigmoid. Adequate preparation impossible due to stenosis.

muscular branches of the inferior mesenteric artery."

On August 26, 1962, after an uneventful postoperative course, the patient was discharged from the hospital.

Third Admission. On October 15, 1962 the patient was admitted to the hospital with symptoms of partial small bowel obstruction. These were relieved by Cantor tube insertion and rest. He was discharged on October 20, 1962 and since then has been asymptomatic.

DISCUSSION

Although infarction with subsequent stenosis of the colon is a rare medical problem, segmental stenosis of the small intestine has been recognized for years as an occasional sequela following repair of a strangulated hernia. More recently, Wolf and Marshak[15] and Rosenman and Gropper[12] have described segmental stenosis of the small intestine following superior mesenteric vascular occlusion. In both instances the small bowel, deprived of its usual blood flow, is partially devitalized and the mucosa, being more vulnerable to ischemia, may slough when ischemia is insufficient to cause permanent damage to the muscularis.[12] Thus the process does not go on to gangrene of the entire intestinal wall with subsequent perforation.

Infarction of the descending colon due to vascular occlusion alone, in the absence of mechanical obstruction, is most uncommon according to McCort.[7] He found only 15 reported cases and added 3 of his own. The infrequency of symptomatic occlusion of the inferior mesenteric artery is borne out also by McClenahan and Fisher[3] who reported a group of over 100 cases of mesenteric artery occlusion of which 96 per cent were in the superior arterial branch.

Infarction of the left colon, however, has been encountered more frequently since the advent of reconstructive surgery of the abdominal aorta. Recently, Young *et al.*[16] reported infarction of the left colon in 8 of 800 persons subjected to abdominal aortic surgery and found 34 other cases reported. Javid *et al.*[6] observed 3 individuals in a series of 660 patients treated for aortoiliac lesions who developed colon necrosis.

None of the reported cases of colon infarction has developed stenosis of the major portion of the descending colon and sigmoid as occurred in our patient. Smith and Szilagyi[14] had 4 patients who developed benign strictures of the rectum, sigmoid or both. Movius[10] also has described 2 patients who developed diaphragm-like rectal strictures about 9 cm. from the anal orifice following surgery on the abdominal aorta. Somewhat similar cases have been reported also by Moore,[16] McKain and Shumacker,[9] and Javid *et al.*[6]

CLINICAL FINDINGS

A clinical picture of hypotension, persistent abdominal distention and diarrhea (which may or may not be bloody) has been found to be highly suggestive of intestinal infarction. Unexplained leukocytosis is additional evidence. If the colon perforates,

shock may develop. Sigmoidoscopy is perhaps the most valuable procedure in the diagnosis of left colon infarction. Initially, the bowel mucosa is cyanotic, later it is edematous with polypoid thickening,[16] and eventually progressive sloughing of the mucosa occurs. Ecchymotic discoloration of the skin about the anus, as noted in our patient, may prove to be an important diagnostic sign. In most of the reported cases, the diagnostic significance of the sigmoidoscopic findings has not been fully appreciated, and it seems likely that, as clinicians become more aware of the diagnostic importance of the mucosal changes in the rectum and sigmoid, earlier diagnosis will become more accurate.

ROENTGEN FINDINGS

Plain Roentgenograms. Roentgenograms of the abdomen reveal a gas pattern consistent with adynamic ileus, usually nonspecific, and tending to increase in severity the first few days following infarction. The thick, edematous, bowel wall may be visualized between gas within its lumen and gas in adjacent loops of bowel. If perforation of the colon occurs, free or trapped extraluminal air may be visible within the abdomen, or if perforation occurs into an adjacent viscus, as the bladder, gas (Fig. 3) may be visible in that structure. As necrosis of the mucosa progresses, bubbles of gas may be visible in the wall of the colon (Fig. 5).

Contrast Studies of the Colon. Soon after infarction, there may be no narrowing of the colon but irritability of the rectum and sigmoid is usually evident. The mucosal folds are wide and flat, or thick and blunt, or at times nodular. Recently, Boley *et al.*[1] described polyp-like thickening of the mucosa along the margins of the colon in reversible vascular occlusion.

As ulceration and sloughing of the mucosa occurs, the barium enema study reveals a total loss of haustral markings and mucosal pattern with tubular narrowing of the lumen (Fig. 6), not unlike that seen in some stages of ulcerative colitis.

In the later stages (Fig. 7 and 8), there is progressive stenosis of the involved colon with marked shortening. Eventually, the bowel presents a string-like appearance. The transition zone between normal and abnormal bowel is cone-shaped, thus differing from the sharp margin and overhanging edges generally seen in carcinoma of the colon.

SUMMARY

A case of sudden infarction of the descending colon and sigmoid due to occlusion of the inferior mesenteric artery in a 62 year old man who was previously well has been presented, with particular emphasis on the roentgen findings. Ultimately, marked contraction, fibrosis and stenosis of the involved bowel required resection and anastomosis of the transverse colon to the rectum. At surgery a small aneurysm of the abdominal aorta was found and probably was the underlying cause of the vascular occlusion. The importance of a sigmoidoscopic examination, if infarction is suspected, and the pathognomonic changes in the colon mucosa are emphasized.

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VOL. 91, NO. 4

THE ROENTGEN FINDINGS OF CARCINOID TUMORS OF THE GASTROINTESTINAL TRACT*

A REPORT OF 12 RECENT CASES

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THE diagnosis of tumors of the intestinal tract presents a challenge to the radiologist.[2,6,9,13] One type of tumor usually not diagnosed until the time of surgical operation is the carcinoid. In 12 patients whose ages ranged from 40 to 80 years, certain characteristics of intestinal carcinoids permitted a correct diagnosis before operation, and these constitute the major reason for this report.

PATHOLOGIC FEATURES

Carcinoid tumors are nonencapsulated, but circumscribed, tumors of the mucosa. They may be of annular or polypoid form. Classically, on transection, they are bright yellow, but may be gray and firm. The tumor consists of small cuboidal cells with nuclei of uniform size and shape.[1] The cells emanate from those described by Kulchitsky, and on staining with silver salts, contain argyrophilic granules. Lubarsch in 1888 recognized these growths as "little carcinomas," but Obendorfer in 1907 differentiated them from carcinomas, naming them carcinoids.

MacDonald[10] suggested that all carcinoid



FIG. 1. Case 2. A 46 year old woman with carcinoid involving the third portion of the duodenum. Oblique view of the gastrointestinal series shows indentation into the lumen of the duodenum (arrows).



FIG. 2. Case 3. Carcinoid of the terminal ileum in a 48 year old man. A filling defect is somewhat irregular in outline. Diverticulitis of the colon is also present.

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Fig. 3. Case 4. Small-bowel barium study in a 52 year old man. Stiff dilated angulated loop of ileum with edematous mucosa and a mass on its upper aspect. The mass is a carcinoid growth, whereas stiffness and edema were produced by the invasion of the mesentery and partial occlusion of veins (arrow).

tumors be considered malignant. When present in the small intestine, invasion into the wall and infiltration of the mesentery lead to fibrosis, angulation of an intestinal loop, and adherence of the peritoneal surfaces. The sharp angulation with a filling defect is recognized as an important roentgen sign of carcinoid tumors. Usually, carcinoid tumors in the ileum metastasize and lead to the carcinoid syndrome; those in the duodenum are usually polypoid lesions.

CLINICAL MANIFESTATIONS

The pharmacologic effects of carcinoid tumors (flushes, chronic diarrhea, respiratory disease, and valvular heart disease) result from the excessive release of serotonin. The metastatic carcinoids elaborate



Fig. 4. Case 6. One hour roentgenogram of a small bowel barium examination in a 65 year old woman. The carcinoid tumor involves the terminal ileum. Several ileal loops are edematous and fixed by tumor invading the mesentery.

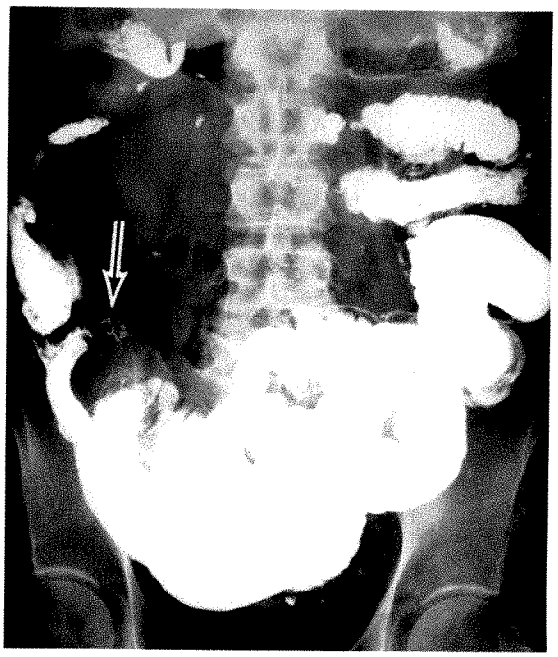


Fig. 5. Case 8. Carcinoid of the terminal ileum (arrow) in a 61 year old man well demonstrated on a roentgenogram of the small bowel.

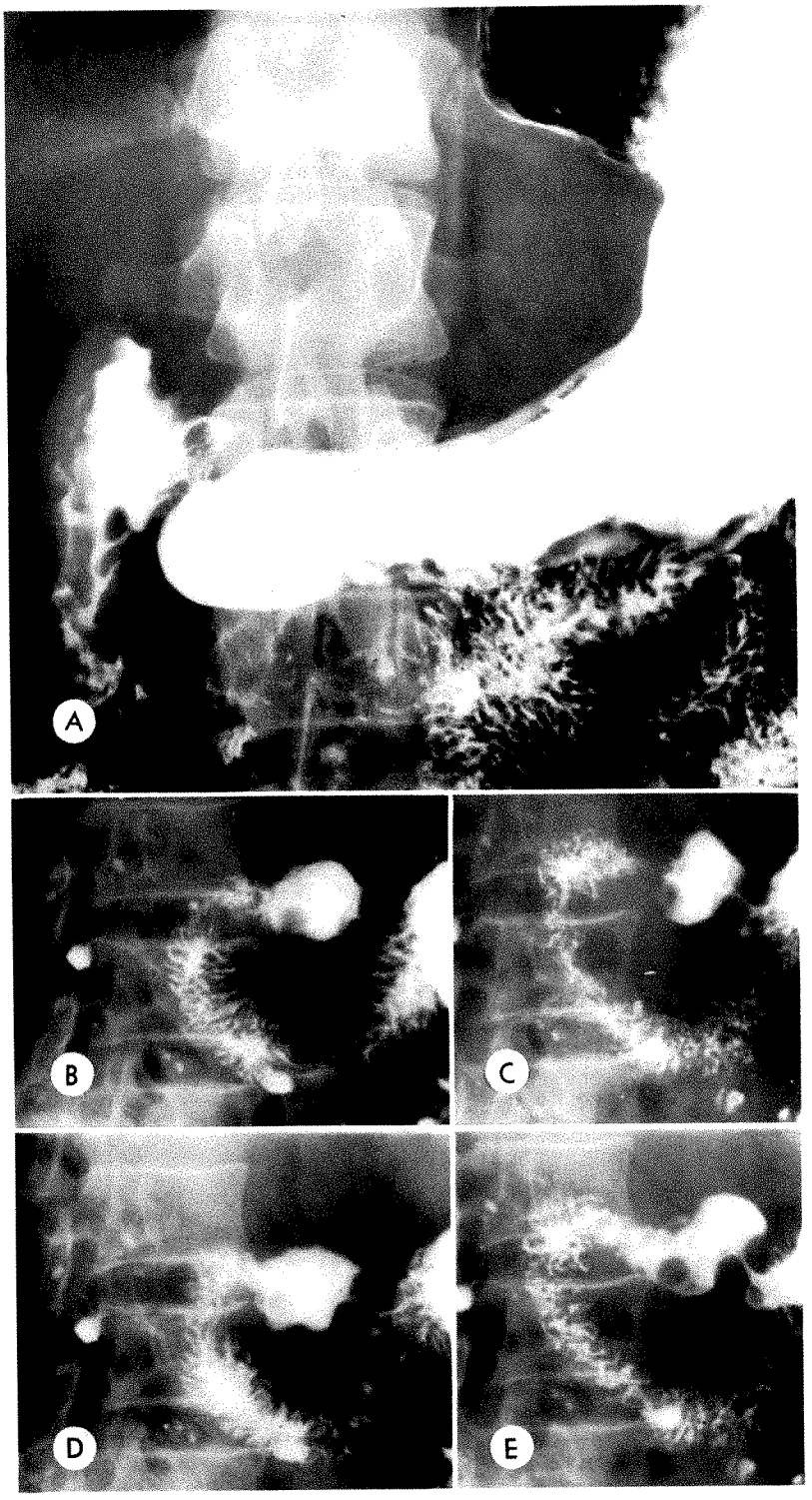


FIG. 6. (*A–E*) Cases 10 and 12. Carcinoid of the duodenal bulb of similar appearance in two different patients. A rounded filling defect was constantly seen.

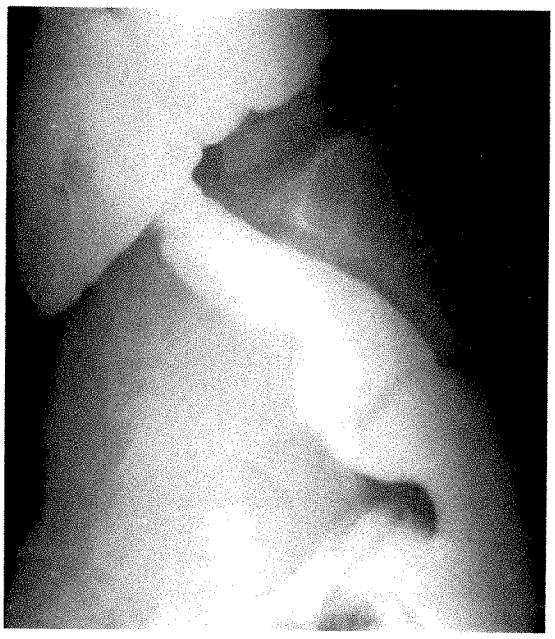


FIG. 7. Case 9. A filling defect in the anterior wall of the rectum produced by carcinoid tumor. This tumor is indistinguishable from carcinoma.

large quantities of serotonin in the form of 5-hydroxytryptamine from the essential amino acid tryptophan.[7-13] It is changed to 5-hydroxyindoleacetic acid which is a urinary metabolite normally present in 24 hour urine specimens in the amounts of 3 to 10 mg. It is present in the urine of patients with this syndrome in amounts of 80 to 800 mg. In patients with metastatic carcinoids a qualitative test for 5-hydroxyindoleacetic acid is positive. The reaction of this test in 5 of the reported patients was positive in 3.

The signs and symptoms of carcinoid tumors are nonspecific, although diarrhea may be suggestive. Patients may complain of intestinal obstruction, hemorrhage, or a palpable mass, but some tumors have been discovered in asymptomatic patients.

Table 1 is a summary of the clinical and roentgenologic findings in 12 patients. Representative roentgenograms illustrate these findings.

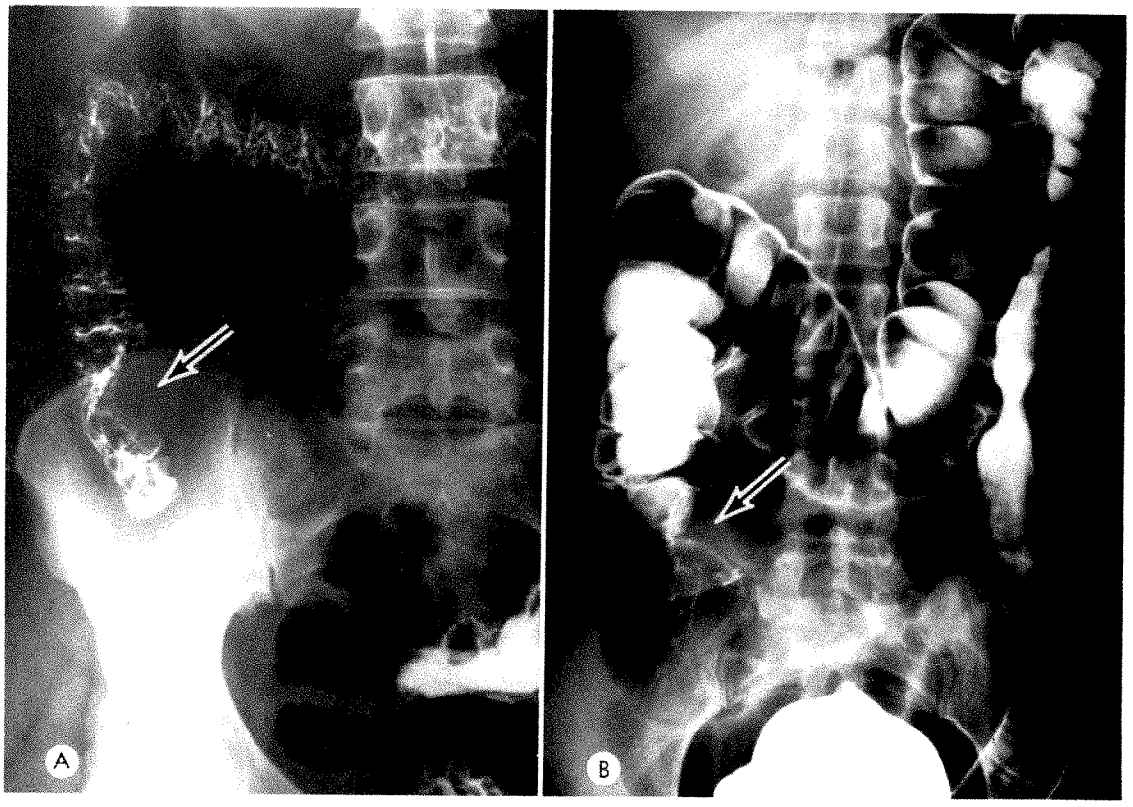


FIG. 8. Case 11. Carcinoid involving the (*A*) ileum and (*B*) ileocecal valve (arrows) in a 40 year old woman. This type of lesion is indistinguishable from primary carcinoma of the ileocecal valve.

TABLE I

CLINICAL AND ROENTGENOLOGIC FINDINGS IN 12 PATIENTS

Case	Age Sex	Symptoms	Signs	Laboratory Findings	Roentgenologic Findings	Surgical Findings
1	63 F	Severe diarrhea, up to 15–20 movements daily	Sea gull murmur, hepatic enlargement	Hb. 13.3 5 H.I.A.A. +	Angulation with filling defect in terminal ileum	Carcinoid of ileum, metastatic; autopsy showed involvement of tricuspid valve
2 (Fig. 1)	46 F	Diarrhea for 3 yr., dyspnea, palpitation, hot flashes	Tachycardia	5 H.I.A.A. +	Partial obstruction of duodenum	Carcinoid of duodenum, metastatic to liver
3 (Fig. 2)	48 M	Melena	Physical examination normal	5 H.I.A.A. −	Filling defects of distal ileum	Carcinoid involving ileum
4 (Fig. 3)	52 M	14 lb. weight loss in 10 mo., left lower quadrant pain	Abdominal tenderness, patient appeared chronically ill	4+guaiac stools, 5 H.I.A.A. −	Stiff loops of ileum with ulceration	Carcinoid of ileum with large lobulated mass
5	54 F	Vomiting	Obstruction		Ileal obstruction	Mass at root of mesentery diagnosed carcinoid
6 (Fig. 4)	65 F	Lower abdominal cramps, diarrhea, 15 lb. weight loss	Abdominal tenderness	Guaiac stools	Irregular lesion of terminal ileum	Carcinoid, metastatic with implants in peritoneum, mesentery, terminal ileum
7	77 M	Right sided pain, nausea and vomiting	Abdominal distention, hepatomegaly	Hct. 40%	Filling defect of terminal ileum	Mass in region of cecum, partially obstructing terminal ileum, metastatic to liver
8 (Fig. 5)	61 M	Vague abdominal pain with recent cramping and diarrhea	Hepatomegaly	5 H.I.A.A. +	Obstruction of terminal ileum	Carcinoid of terminal ileum, metastatic to liver
9 (Fig. 7)	42 F	Nausea, vomiting	Abdominal tenderness		Polypoid filling defect in rectum	Carcinoid in polyp, salpingitis
10 (Fig. 6A)	80 M	Diarrhea	Not remarkable	Guaiac stools	Upper gastrointestinal polypoid defect of duodenal bulb (barium enema study read as diverticulosis)	Carcinoid in polyp of duodenum

TABLE I (*Continued*)

Case	Age Sex	Symptoms	Signs	Laboratory Findings	Roentgenologic Findings	Surgical Findings
11 (Fig. 8, *A* and *B*)	40 F	Gas, right lower quadrant pain	Distention, right lower quadrant tenderness	Hb. 8.6	Filling defect involving cecum and ileocecal valve	Carcinoid of ileocecal valve
12 (Fig. 6, *B–E*)	44 M	Abdominal pain with meals	Unremarkable		Intramural filling defect of duodenal bulb	0.9 cm. nodule in duodenum, carcinoid

DISCUSSION

According to Dockerty,[5] 90 per cent of carcinoid tumors occur in the appendix. Metastatic spread to local lymph nodes has been reported in 3.5 per cent of 825 patients. Tumors in the appendix are rarely detected by roentgenologic examination. In reviewing 149 cases of carcinoid tumors in locations other than the appendix, MacDonald[10] found 74 in the ileum, 17 in the jejunum, 27 in the large bowel, 14 in the stomach, and 8 in the duodenum. The remainder were in various other locations. This general distribution correlates well with the experience of others.

The examination of the small intestine is complicated by its length of 22 feet, which constitutes 75 per cent of the gastrointestinal tract. Examination, however, is facilitated with neostigmine sulfate as described by Margulis and Mandelstam.[11] Our routine is to obtain roentgenograms of the small intestine during all examinations of the upper gastrointestinal tract.

Horsley and Golden,[8] in a review of the world literature in 1957, reported 17 cases of authenticated carcinoid tumors of the duodenum and added 2 of their own. Another case was reported in 1960 by Costello and Aitken.[3] In our series, polypoid defects involved the first duodenum in 2 patients and partially obstructed the third duodenum in another patient.

The most distinctive feature of carcinoid of the small intestine, in our experience, is the multiplicity of areas involved in the bowel. Except for polyps, no other lesions show this multiplicity. If roentgenograms show kinking of the small bowel, as in several of our cases, the mass causing it is even more likely to be a carcinoid. Polypoid lesions in the duodenum and rectum were nonspecific.

DIFFERENTIAL DIAGNOSIS

In tumors of the bowel, carcinoma must be considered in the differential diagnosis. This disease is more commonly situated in the proximal small bowel, and is associated with a higher incidence of obstruction. Carcinoma tends to extend over a single short segment of the bowel. A lymphoma usually involves the small bowel more extensively than does carcinoid and, as a rule, is not associated with kinking of the bowel. In a review of patients with lymphoma, Darling and Welch[4] reported that 50 per cent had annular lesions. The intestinal mucosa is corrugated, and often the areas of lymphomatous invasion are extensive. With carcinoma of the ileocecal valve, the mass is larger and usually ulcerated.

Another disease to be considered in the differential diagnosis is regional ileitis. The long, narrow, rigid segments of bowel as seen in regional enteritis were not encountered in our series of cases of carcinoid.

Carcinoids in the duodenum are not distinctive and may simulate adenoma, myoma, pancreatic rest, lipoma, or prolapsed mucosa.

SUMMARY

Gastrointestinal carcinoids in 12 patients are described. Three of the 12 had the carcinoid syndrome. The tumor was found in the

duodenum in 3. Carcinoid should be suspected if roentgenograms show multiple lesions in the ileum, particularly with buckling and angulation of the bowel.

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PLEXIFORM NEUROFIBROMATOSIS OF THE COLON SIMULATING HIRSCHSPRUNG'S DISEASE*

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NEUROFIBROMATOSIS is a disease with multiple manifestations. We have observed a patient with plexiform neurofibromatosis of the large bowel whose clinical and roentgenographic findings simulated those present in Hirschsprung's disease.

REPORT OF A CASE

This 8 year old white male (birth date April 10, 1955) was first seen in the St. Louis Children's Hospital on October 14, 1959 with complaints of chronic constipation, gigantism of the right lower extremity particularly marked in the foot, and multiple large subcutaneous masses involving the back, buttocks, and right lower extremity. Between the ages of 8 months and 4 years, he underwent multiple surgical procedures for excision of recurrent subcutaneous masses of the foot and back. Bilateral inguinal hernias were repaired at 31 months of age.

The child was a product of a normal pregnancy and delivery. There was mild mental retardation. Bowel movements had always been assisted by the use of cathartics and enemas.

Physical examination demonstrated a small, somewhat malnourished child with the right leg markedly larger in diameter than the left, due to multiple soft tissue tumors. The right fourth and fifth toes with their metatarsals were absent as a result of previous surgery. The abdomen was protuberant. Superficial venous distention was noted in the epigastrium. There were bilateral herniorrhaphy scars and a large soft tissue mass in the right posterior subcostal area at the site of a previous tumor excision. Several additional large tumors were noted on the left back and right buttock. A mild dorsolumbar scoliosis was present. No café au lait spots were seen.

Barium enema studies (Fig. 1, *A* and *B*) demonstrated narrowing of the rectum and of the distal portion of the sigmoid colon. There was abrupt transition in the caliber of the lumen in the lower sigmoid. The large intestine proximal to this point of transition was redundant and dilated. Hyperperistalsis was evident in the sigmoid above the transition point. A considerable amount of stool was present proximal to the area of narrowing. On the postevacuation roentgenograms, a sizable residue of barium persisted within the colon. The findings were felt to be consistent with Hirschsprung's disease.

On November 25, 1959 frozen section rectal biopsies showed no ganglion cells. Consequently, a Swenson pull-through procedure[41] was performed. The distal 10 cm. of sigmoid colon was resected along with most of the rectum. Frozen sections demonstrated an adequate number of ganglion cells at the proximal aspect of the resected segment. On permanent sections, however, ganglion cells were present throughout the entire specimen along with enlargement and increase in number of the nerves of the submucosal and myenteric plexuses (Fig. 2, 3 and 4). There was short lived improvement in evacuation of the colon following surgery.

On August 30, 1960 a large mass on the right back and buttock, measuring 12×10×6 cm., was resected. Histologic examination showed mature fat with large and small nerves coursing through it. The diagnosis was hypertrophy of fat and nerves, neurolipomatosis. The previously resected tumors of the foot and back had the same microscopic appearance.

Because of continued difficulty in defecation during the 20 months following the Swenson procedure, a barium enema examination was done on July 20, 1961. It demonstrated narrowing in the remaining rectum and distal colon with proximal colonic dilatation. Evacuation, however, was improved over that in 1959.

By August 10, 1962, 33 months following surgery, the child was experiencing more difficulty passing stool and daily enemas and cathartics were only of limited effectiveness. A barium enema study at this time showed an area of narrowing in the distal colon similar to the previous study (Fig. 5). Barium sulfate administered orally was not handled well by the colon and accumulated above the narrowed

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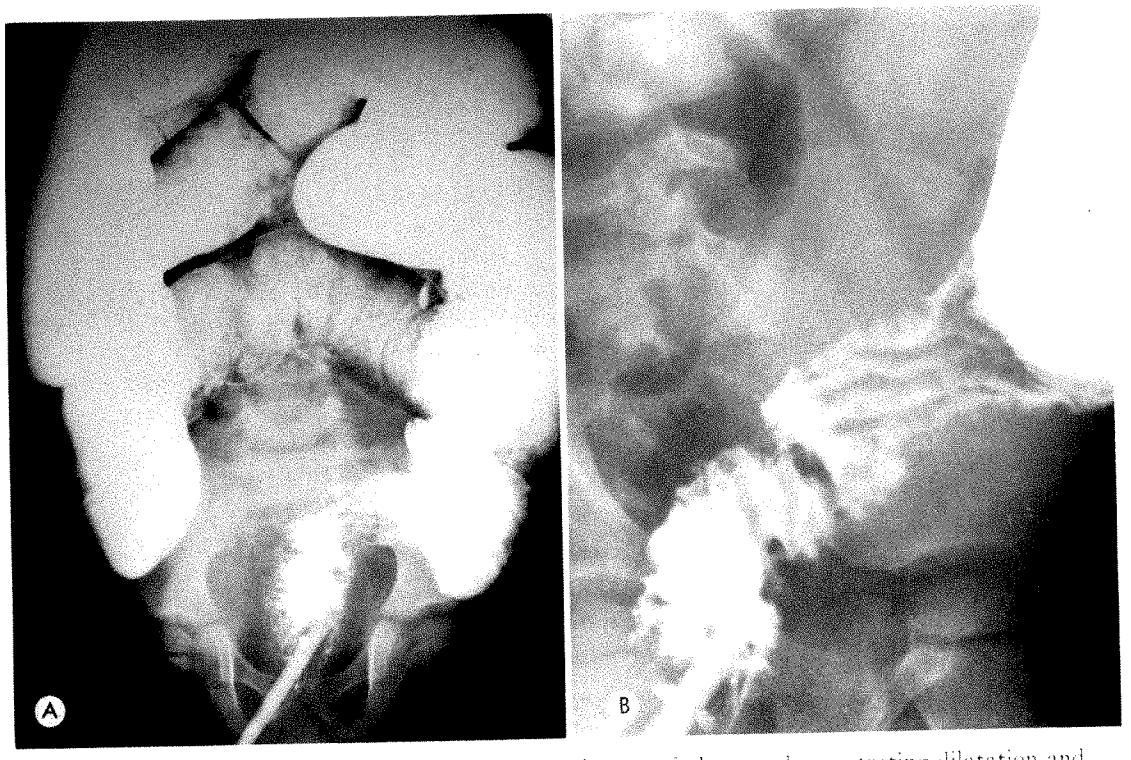


FIG. 1. (*A* and *B*) Initial barium enema study at the age of 4½ years demonstrating dilatation and redundancy of the colon with narrowing of the rectosigmoid.

segment, although evacuation aided by Lavema* at the time of the barium enema examination was satisfactory.

On August 11, 1962 biopsies were taken 6 cm. from the anal verge. Examination of this material and review of the previous sections were interpreted as representing neurofibromatosis of the rectum and colon and it was suggested that a resection be carried out to an area in which abnormal nerves were no longer present. On August 24, 1962 the child underwent celiotomy and plexiform neurofibromatosis was found on biopsy in the sigmoid colon. Serial biopsies were taken up to the mid-transverse colon at which point frozen sections suggested a decrease in the extent of involvement by abnormal nerves. Subsequent resection of the left transverse, the descending, and the remaining sigmoid colon was carried out and the right portion of the transverse colon was anastomosed to the anal verge (Fig. 6).

Histologic examination of the resected colon demonstrated the nerves of the myenteric and submucosal plexuses to be increased both in size and number. The appearance resembled that in the previously resected sigmoid colon



FIG. 2. A representative section through the colon. Located between the circular and longitudinal muscle layers is a group of enlarged nerves of the myenteric plexus (arrows) (H. & E. ×33).

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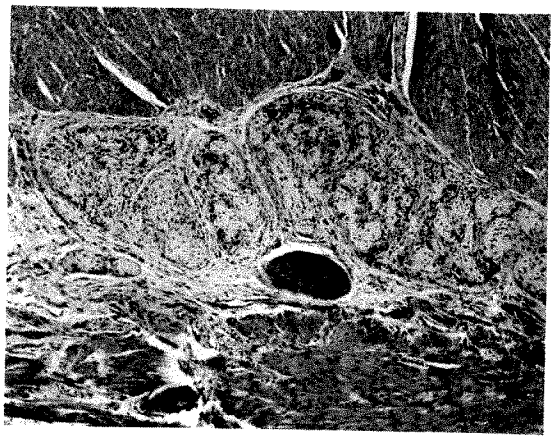


FIG. 3. A higher magnification demonstrating an increase in number and enlargement of the nerves in the myenteric plexus (H. & E. ×85).

and rectum. There was no invasion of muscle or surrounding tissue by nerve elements. Ganglion cells were present in normal numbers throughout. Although abnormal nerves varied somewhat from section to section, it was felt that there was no tendency toward decreased involvement at the proximal end of the specimen, as was initially suggested on the frozen sections.

An ascending colostomy done at the time of the colonic resection was closed on September 29, 1962. On October 8, 1962 a wound abscess was drained. On November 1, 1962 he was again admitted for repair of recurrent bilateral inguinal hernias and varicoceles. A 2×3 cm. mass was noted at the inferior aspect of the colectomy scar. This was resected and on histologic examination demonstrated a cavernous hemangioma in addition to foreign body reaction from the suture. The two resections of the large bowel have thus far been ineffectual in alleviating the constipation.

DISCUSSION

Neurofibromatosis is primarily characterized by subcutaneous nodules, skin lesions, and various neurogenic tumors.[43] To our knowledge this is the first reported instance of plexiform manifestation of these tumors in the colon. Of particular interest is the close resemblance, both clinically and roentgenographically, to aganglionic megacolon.

The bony lesions of neurofibromatosis have been described.[23] These include scoliosis of varying severity, defects of the walls of the orbits, erosive defects caused by adjacent neurogenic tumors, bowing and pseudoarthrosis of the lower leg and disorders of growth of bone associated with elephantoid hypertrophy of the soft tissues. The only abnormal manifestation in this patient was elephantoid enlargement of the right leg and foot due to overgrowth of soft tissue tumors. The subcutaneous nodules noted on this patient were neurolipomata made up primarily of mature fat with enlarged nerve fibers. The association of subcutaneous lipomatous masses in patients with neurofibromatosis has been observed.[1,39]

The presence of neurofibromata within the gastrointestinal tract has been reported on numerous occasions.[5,8,9,15,17,19,26,29,36,37,41,46] These lesions may or may not be associated with classic neurofibromatosis.[5] Neurofibromata present themselves as submucosal tumors or tumors scattered throughout the distribution of the splanchnic nerves. The small bowel, appendix, and the stomach are involved most often. Fewer lesions have been reported in the colon and rectum.[2,9,18,20,24,40] All of those lesions previously reported demonstrated localized tumors in one or more areas in contrast to the present case of diffuse plexiform involvement.

The majority of gastrointestinal neurofibromata are asymptomatic. There may be gastrointestinal bleeding, occasionally massive. Rare instances of intussusception and/or perforation have been reported.[5,8,17]

Plexiform neurofibromatosis was originally described as masses of nerves within

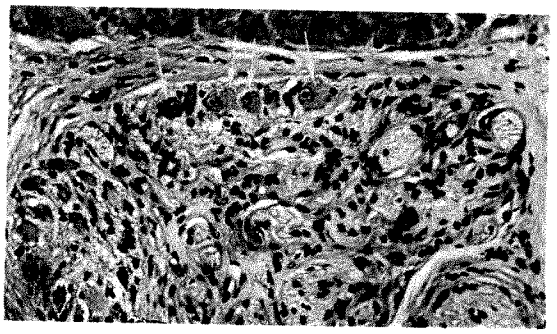


FIG. 4. Normal appearing ganglion cells (arrows) were associated with the groups of enlarged nerves throughout the colon (H. & E. ×340).

the subcutaneous tissues, particularly in the extremities.[14] Further enlargement of these masses with proximal growth toward the central nervous system and eventual death by encroachment upon the spinal cord was commented on.[14,27] Cases have been reported since involving various areas including the pharynx,[4,11,16,28] tongue,[3] maxillary antrum,[35] orbit and globe,[12] neck,[30,32] heart,[32] urinary bladder,[13,45] trigeminal nerve,[31] ear and nose.[33]

The presence of Hirschsprung's disease[22] may be manifested from the first day of life or may not be diagnosed until many years later. The typical story is one of chronic constipation with varying degrees of abdominal distention. There may be neonatal congenital megacolon, often presenting as delay in passage of meconium, early vomiting, and abdominal distention. Enterocolitis and/or exudative enteropathy may be seen.[6] Obstruction may be partial,



Fig. 6. Barium enema study after resection of the distal one-half of the colon and anastomosis of the right transverse colon to the anal verge. Dilatation of the colon persists.

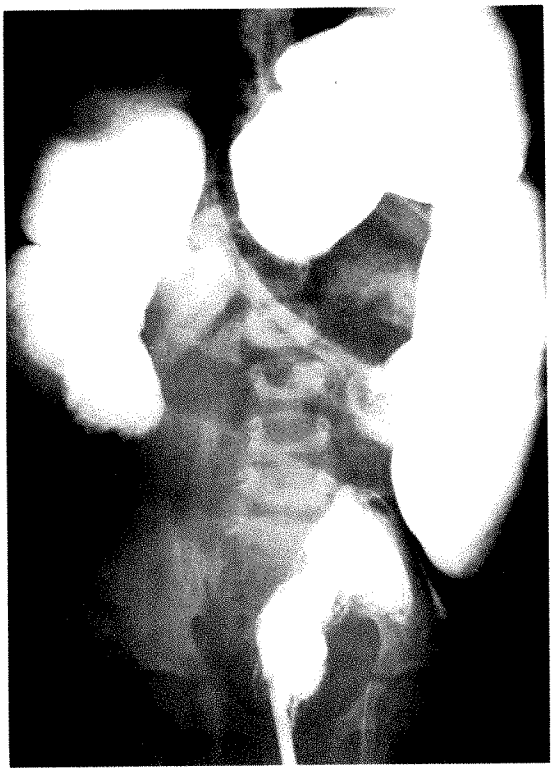


Fig. 5. Barium enema study on August 10, 1962 at the age of 6 years, 10 months, demonstrating almost identical findings as those seen on the initial examination.

intermittent, or complete. Early episodes of obstruction may be relieved by passage of stool either spontaneously or with the aid of an enema.[7,25,34,38] Intermittent diarrhea may confuse or delay the establishment of the correct diagnosis at any age.

The roentgen diagnosis is based primarily on the barium enema examination demonstrating a narrowed poorly distensible distal large bowel segment with dilatation of the colon proximal to the involved area and poor evacuation of the colon. An uncleansed large bowel helps to accentuate the difference between the dilated proximal segment and the narrowed distal segment. A defunctionalized segment following colostomy will tend to obliterate the area of transition. Delayed postevacuation lateral roentgenograms of the rectum have been quite helpful in the diagnosis of questionable cases. The characteristic proximal dilatation may not be present in the neonate or during early infancy. Residual barium sulfate in the bowel 1 and 2 days after diagnostic studies is strongly suggestive of Hirschsprung's disease.

The narrowed distal aganglionic bowel

and the proximal dilated bowel filled with either fecal material, air, or fluid may be observed on the plain roentgenograms of the abdomen. In the neonate because of the difficulty in distinguishing large from small bowel, distal small bowel atresias and meconium ileus may be confused on the abdominal roentgenograms. The barium enema study should differentiate these conditions as well as anal stenosis, meconium plug syndrome,[11] and functional megacolon.

The definitive diagnosis is made by histologic examination of the involved large bowel showing the absence of ganglion cells in the myenteric plexus. Swenson *et al.*[42] have described a method of biopsy of the muscular wall of the rectum. An adequate specimen of muscle is not always obtained. Bodian (quoted by Langer and Thomson[25]) biopsied the rectal mucosa looking for changes in the submucosal plexus similar to those seen in the myenteric plexus. The changes in the latter method may be difficult to interpret. Hiatt[21] recommends a posterior approach to the rectum with small transverse incisions midway between the anus and coccyx in order to get a more satisfactory specimen of muscle wall without entering the rectal lumen.

In the child under consideration, the roentgenographic findings were similar to Hirschsprung's disease as there was a narrowed segment of rectum and sigmoid with proximal colonic dilatation and poor evacuation of the contrast material. A rectal biopsy on frozen section showed no ganglion cells; however, the permanent sections demonstrated the presence of normal appearing ganglion cells with an increase in size and number of the associated nerves. The exact relationship between the abnormal nerves and function of the colon is unknown.

The initial surgical treatment in this patient was resection of most of the rectum and a portion of the sigmoid. This did not relieve the constipation. It was elected to resect a greater portion of the colon with a view toward removing the involved bowel up to that point at which the hypertrophied nerves became fewer in number. This point was suggested in the mid-transverse colon by frozen sections but was not confirmed on permanent preparations. To date, there has been little change in the degree of constipation following removal of the distal one-half of the colon.

SUMMARY

A case of plexiform neurofibromatosis of the colon simulating Hirschsprung's disease is reported.

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THE PNEUMATIC COMPRESSION PADDLE IN COLONIC ROENTGENOGRAPHY

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IN GASTROINTESTINAL roentgenologic procedures, compression techniques are acknowledged as a *sine qua non* for the detection and demonstration of lesions not otherwise disclosed. The coned attachment of the serial spot filmer is the most commonly used compression device. Manual manipulation with the lead protected gloved hand is also utilized to a great extent and offers ease of graded compression. Localized pads of nonopaque material, when used for this purpose, do not lend themselves well for grading the compression.

In the past many authors have stressed the value of a pneumatic paddle for compression purposes in studying the upper gastrointestinal tract and the gallbladder with its ducts.[1–4] This report will concern itself with the use of such a pneumatic compression paddle as a means of disclosing and documenting lesions in the colon.

The pneumatic compression device, shown in Figure 1, *A* and *B*,* has been successfully employed for purposes of delineating colonic lesions not otherwise demonstrable. It consists of a banjo like frame made of five ply plexiglass with a round housing and sturdy handle. An inflatable rubber bladder is installed in the round housing with an inflation controlling bulb at the end of the handle. The handle is notched to provide a nonslip grip. Imbedded in the round plexiglass housing is a lead circle which surrounds the inflatable bladder and clearly shows fluoroscopically the region under compression. The inflation controlling bulb permits grading of the compression. The back of the paddle has a rubberized cork facing so it will not slip on the roentgenographic table once it is in position.



FIG. 1. (*A*) Frontal and (*B*) profile views of the pneumatic compression paddle with its rubber bladder partially inflated.

Because of the flexibility of the compression paddle, it can be used anywhere on the recumbent abdomen including the costal cage. Its best application is made with the patient prone, but it can also be used with the patient supine between the locked fluoroscopic screen and the patient.

METHOD OF USE

In positioning the paddle to the area of interest, the device should be grasped close to its neck since this permits more accurate

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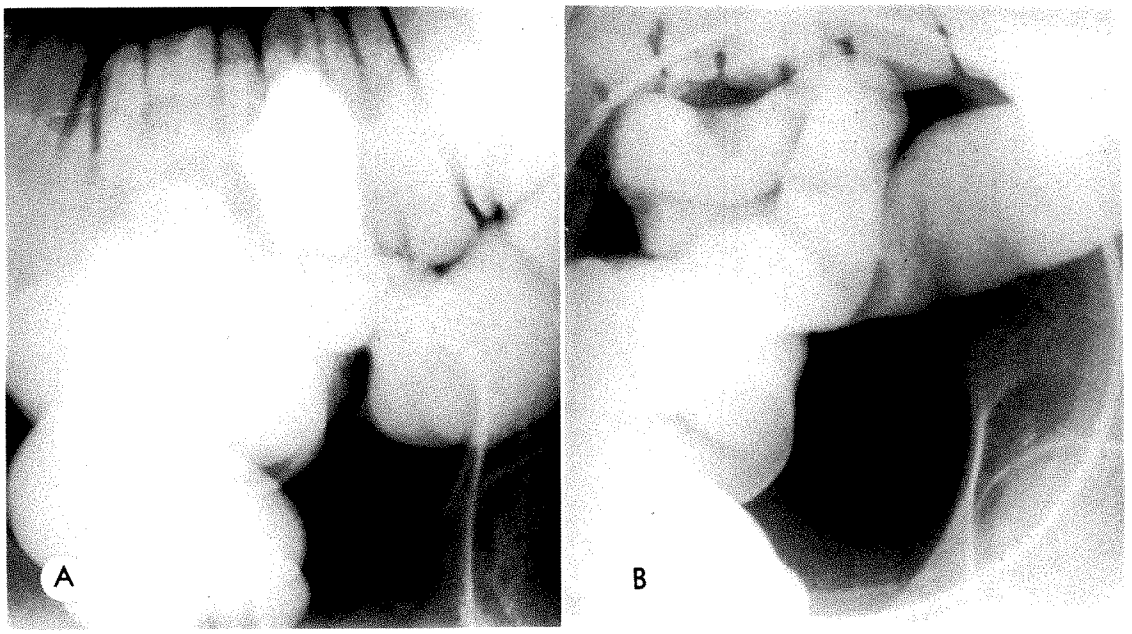


Fig. 2. (*A*) Redundant overlapping sigmoid colon with the transverse colon also superimposed on it. (*B*) Compression paddle separates and thins these loops for adequate visualization and diagnosis.

initial positioning. For minor adjustments thereafter, the paddle can be manipulated with its notched handle. The lead ring surrounding the bladder will guide the fluoroscopist to the colonic segment under study, and rotation or movement of the patient to an optimum position can be accomplished with the paddle in place and the bladder deflated. Movement over the inflated rubber bladder will tend to pull it out of the opening of its housing. Initially, the bladder can be overinflated, and then with gradual deflation or reinflation, the proper amount of graded compression to demonstrate the lesion can be attained. Under table spot roentgenograms are made in the optimum phase of respiration for showing the lesion, or the prone patient can be left centered in position with the paddle in place for over table roentgenography.

EXAMPLES OF USE

Accompanying figures show the value of the pneumatic compression paddle for the demonstration of lesions not otherwise shown or not shown to an optimum degree (Fig. 3, *A*, *B*, and *C*, through 8, *A* and *B*). In the pelvic portion of the colon (including the iliac and sigmoid segments) marked redundancy with overlapping of loops represents the best indication for the use of this device (Fig. 2, *A* and *B*). In some patients the transverse colon dips down to overlay the pelvic colon (Fig. 2*A* and 8*A*), and in others a very long mobile cecum may obscure the pelvic colon. When spillover has occurred into the small intestine, the graded compression applied to the pelvic portion of the colon and the barium filled small intestine often separates these loops so that proper study can be made.

The flexures are usually inaccessible to direct compression with a cone, but with the patient prone, the compression paddle does give rise to some fixation and thinning of the part so that it is very valuable in the demonstration of lesions of the flexures. It is quite important that the proper phase of respiration be employed when studying the flexures, since deep inspiration occasionally permits the flexures to drop below the costal cartilages of the rib cage.

The ascending and descending portions

of the colon, as well as the transverse portion, can often be adequately compressed without this device, but for purposes of immobilizing a polypoid lesion that freely floats on a pedicle, "transfixation" by means of the pneumatic compression paddle not only stills the part but also brings into sharp relief the lesion under study (Fig. 4*B* and 5*B*).

In the cecum the paddle is an extremely important adjunct. It helps differentiate ileocecal valve lips, polypoid tumors, and inverted appendiceal stumps (Fig. 7, *A* and *B*).

CONCLUSION

The pneumatic compression paddle provides guided graded compression studies of

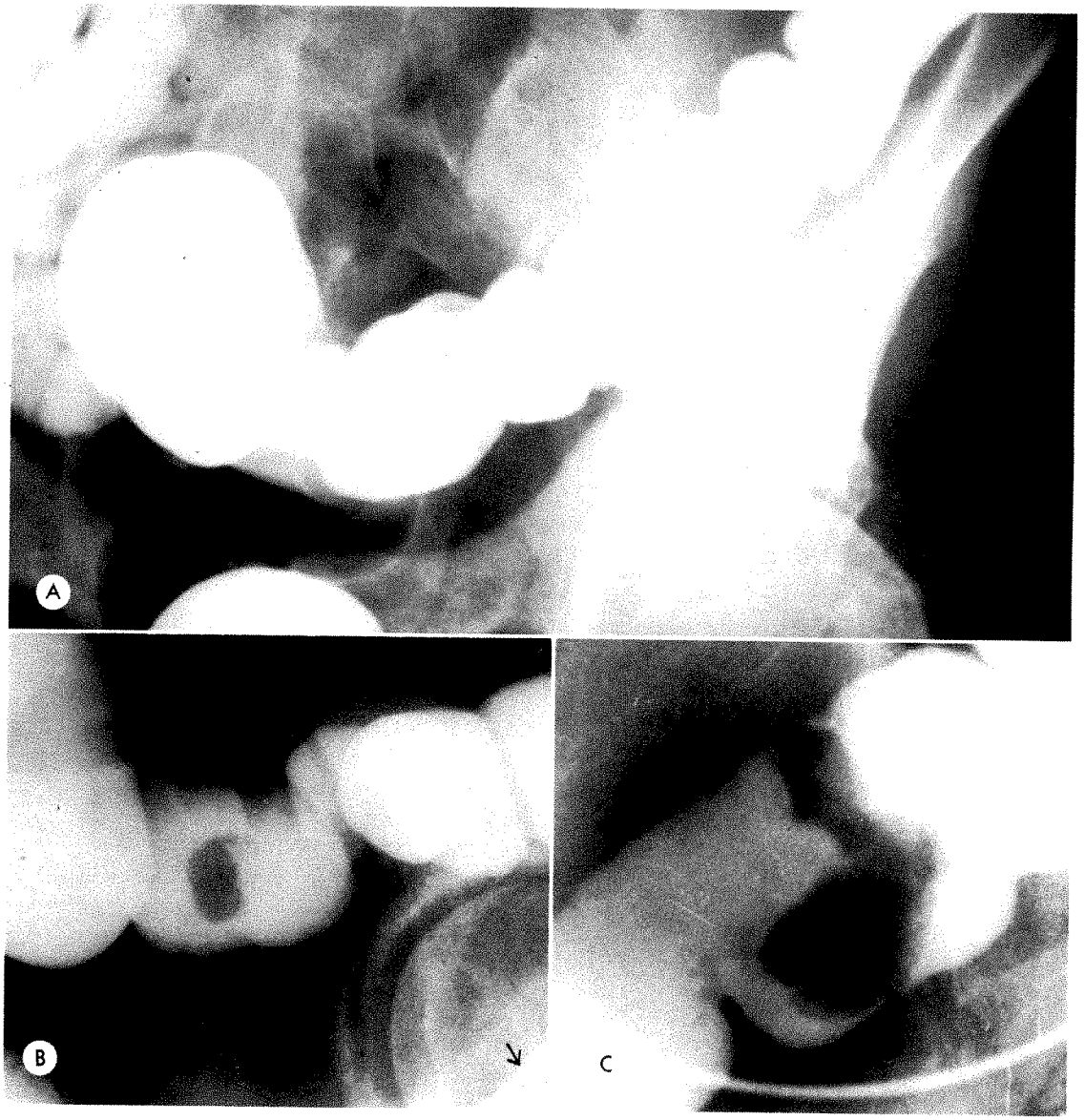


FIG. 3. (*A*) The sigmoid and iliac portions of the colon both harbor pedunculated polypoid lesions which are not shown without the benefit of graded compression. (*B* and *C*) Compression with the paddle thins the part and stills it, thus "transfixing" the pedunculated lesions for documentation. Arrow in *B* points to the buried lead circle in the paddle for proper fluoroscopic placement.

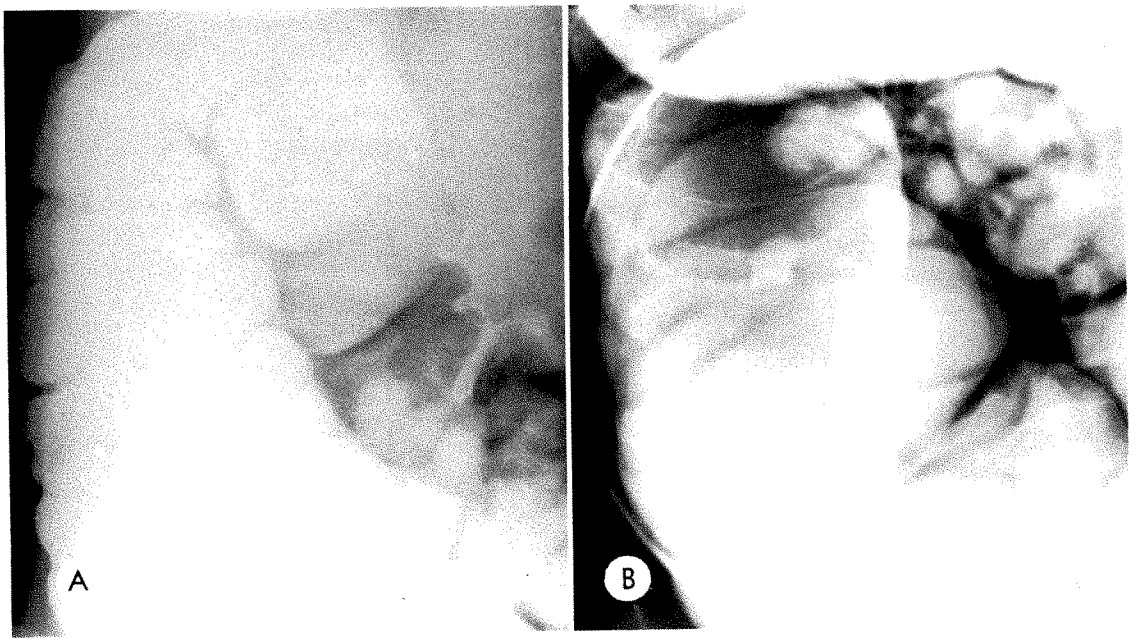


Fig. 4. (*A*) The ascending colon spot roentgenogram fails to show the polypoid lesion in it even though high kilovoltage and lucent type barium have been used. (*B*) With the pneumatic compression paddle properly positioned and inflated so as to thin and fix the part, the lesion is well delineated.

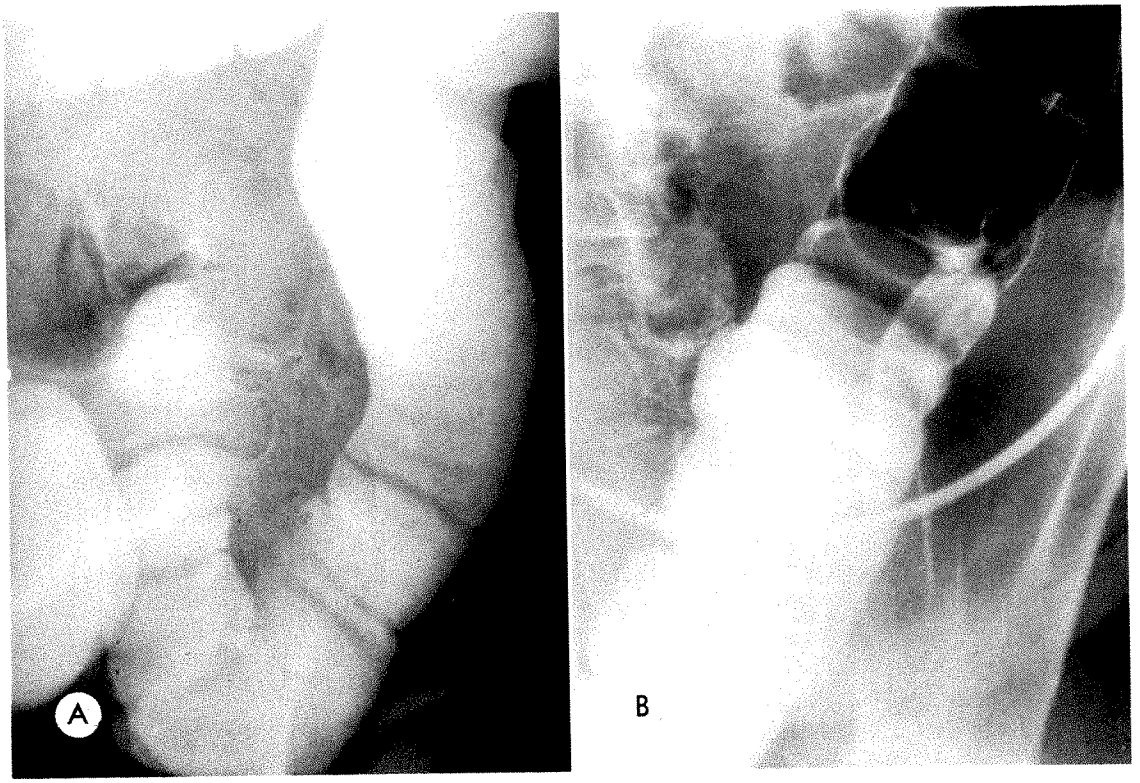


Fig. 5. (*A*) The pneumocolon spot roentgenogram of the distal descending and iliac portions of the colon appears normal, though it contains a free floating stalked polypoid lesion. (*B*) With graded compression, the long stalked polypoid lesion is clearly defined.

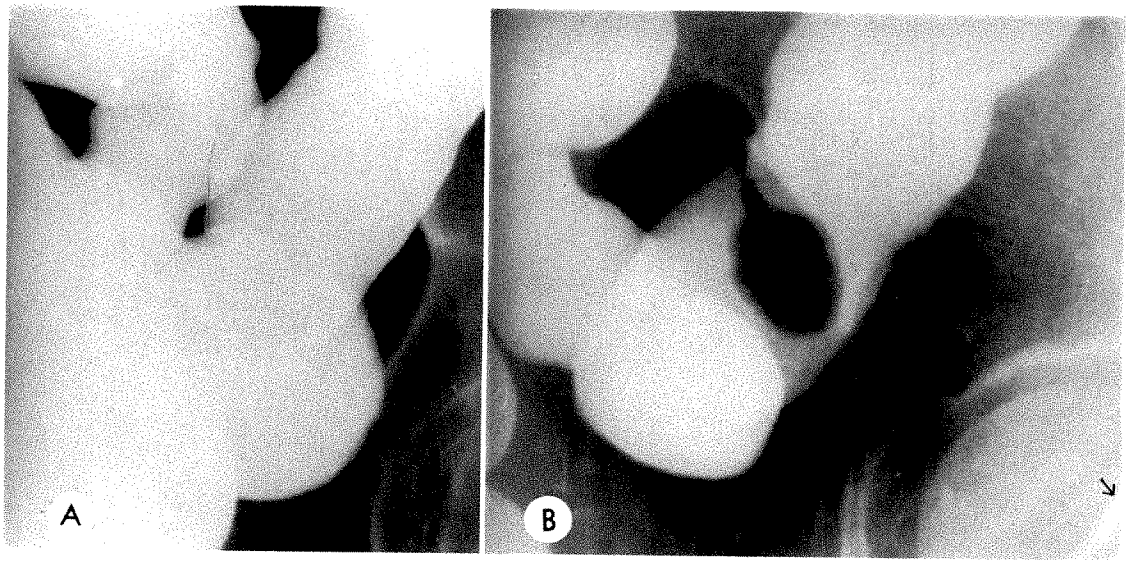


Fig. 6. (*A*) The colon at the brim of the pelvis appears normal on a roentgenogram made with high kilovoltage technique. (*B*) The pedunculated polypoid lesion in this region now stands out sharply because it is fixed by the compression paddle. Arrow points to the lead ring surrounding the rubber bladder.

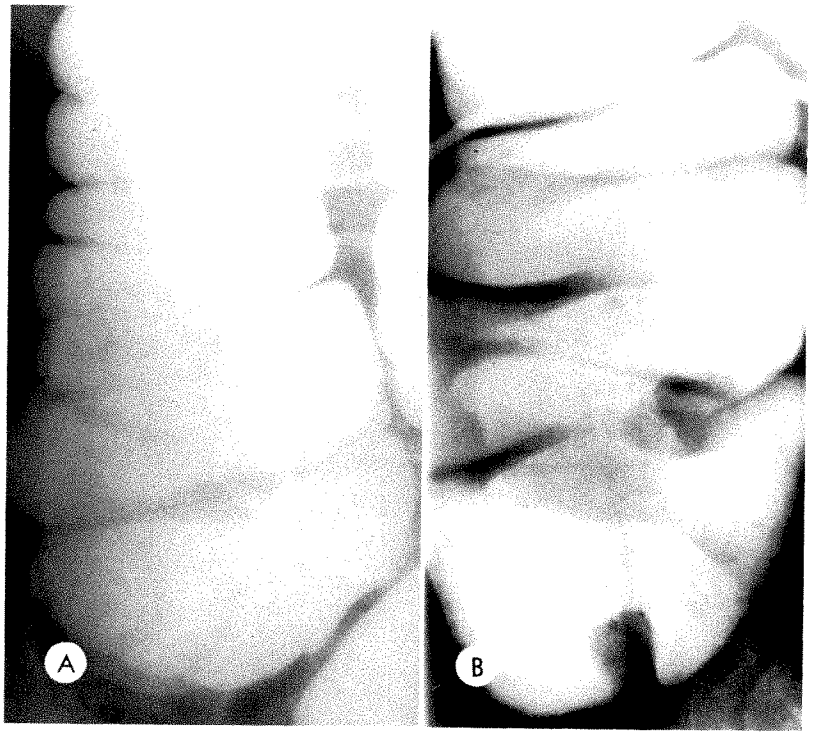


Fig. 7. (*A*) The cecum without compression does not show the rather large inverted appendiceal stump. (*B*) With the pneumatic compression paddle, the granulomatous lesion which was ulcerating and was a source of anemia is clearly visualized.

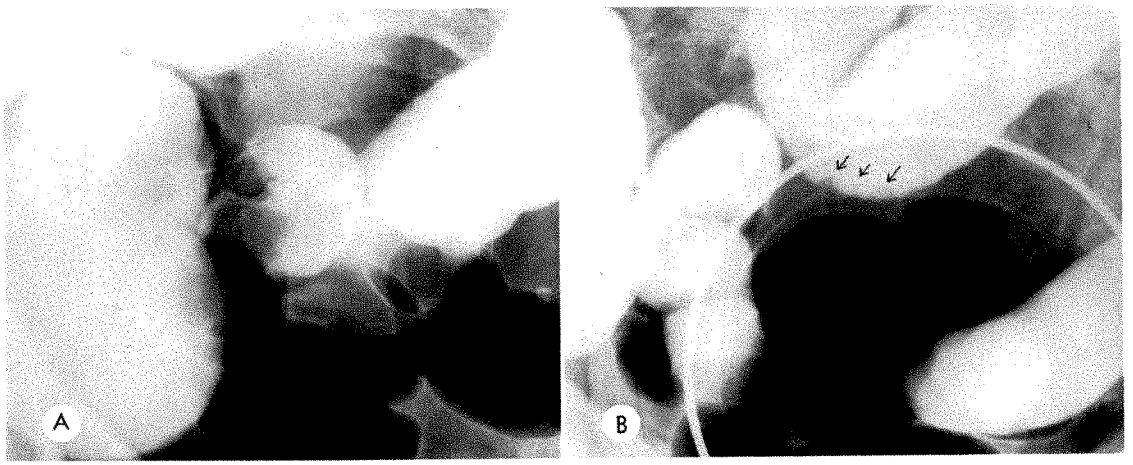


FIG. 8. (*A*) The pneumocolon spot roentgenogram shows the low-lying transverse colon overlapping the sigmoid. (*B*) The pneumatic compression paddle has displaced the mid-transverse colon cephalad and apart from the sigmoid to show a flat villous adenoma (arrows) on the lower margin of the mid-transverse colon.

the colon and is a most valuable adjunct in the demonstration of lesions often otherwise not shown. The device not only immobilizes and thins the part but effectively displaces overlapping intestinal loops that often prevent proper disclosure and demonstration of colonic lesions.

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INTRACOLONIC PRESSURES DURING BARIUM ENEMA EXAMINATION*

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WHILE performing barium enema examinations, the author has often wondered whether the barium can safely be raised above 3 feet when the flow is slow. The solution of this problem depends on the answers to the questions, "What is the pressure tolerance of the colon?" and "What pressures are developed when a barium sulfate suspension is introduced into the colon?" To obtain these answers 4 types of experiments were carried out.

EXPERIMENT NO. 1

COMPARISON OF PERFORATING TENSIONS OF LIVING AND DEAD DOG INTESTINE

While the pressures required to perforate segments of human intestine at autopsy have been known for some time,[1] it is recognized that there is an argumentative leap in extrapolating data from postmortem intestines to living human intestine. In an effort to bridge this leap, segments of intestine were perforated in 3 dogs, anesthetized with sodium pentothal, by injecting 50 cc. increments of air at a rate of about 5 cc. per second; the air was injected into a Y-tube that connected a Bardex balloon catheter in the tested segment of intestine, with a mercury manometer. The volumes of air and the intraballoon pressures at the times of perforation of the muscularis and the mucosa were recorded (Fig. 1, *A* and *B*); the pressure transmitted to the gut wall was calculated by subtracting the intraballoon pressure for a given volume of air with the balloon out of the intestine from the intraballoon pressure for the same volume of air when the balloon was in the intestine. This subtraction, described by Nathan and Kohen[9] yields a difference that is the pressure exerted by the gut wall against the balloon in the intestine. Since action equals reaction, this pressure exerted by the gut wall against the balloon is equal to the pressure transmitted by the balloon through the gut wall.

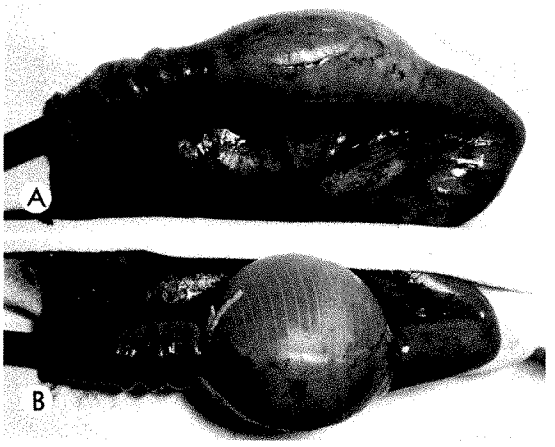


FIG. 1. (*A*) Perforation of intestine occurs in two steps. In this photograph the first step, a longitudinal split of the serosa and muscularis is highlighted. (*B*) With more distention the mucosa ruptures. Human intestine perforates in the same manner as this specimen of dog gut.

The 3 dogs were sacrificed, and the procedure was repeated on adjacent segments of jejunum, ileum, and colon at intervals of 2, 4, and 6 hours after death. The circumference of each perforated segment was measured; then the calculated radius was multiplied by the calculated pressure transmitted through the gut wall to determine the tension in the segment at the time of perforation of the muscularis and the mucosa. This application of the LaPlace theorem to cylinders of tissue is discussed by Burton,[2] and it is expressed by the formula: $T = PR$ (Fig. 2). The tension, or force, tending to tear open the cylinder on either side of an imaginary slit in the wall is the

* Work done at the Department of Radiology, Indiana University Medical Center, Indianapolis, Indiana, and the U. S. Veterans Administration Hospital, West Tenth Street, Indianapolis, Indiana.

product in dynes of force per centimeter length of slit when the transmural pressure in dynes per square centimeter is multiplied by the radius in centimeters.

These calculated tensions, in thousands of dynes per centimeter, are compared (Fig. 3) rather than transmitted pressures or balloon volumes because tensions take into account the radii in addition to the transmural pressures. Although there is considerable variation, there is no consistent difference in the tolerance of dog intestine, with the animals alive or dead up to 6 hours.

The intestines of 4 other dogs were perforated with the dogs under anesthesia or dead up to 6 hours, but in these 4 dogs the balloon volumes or the intermittent pressures obtained without a Y-tube were measured. In these dogs also, no real difference was found between adjacent segments of intestine whether alive or dead. Because of the difference in method, these data could not be included in Figure 3.

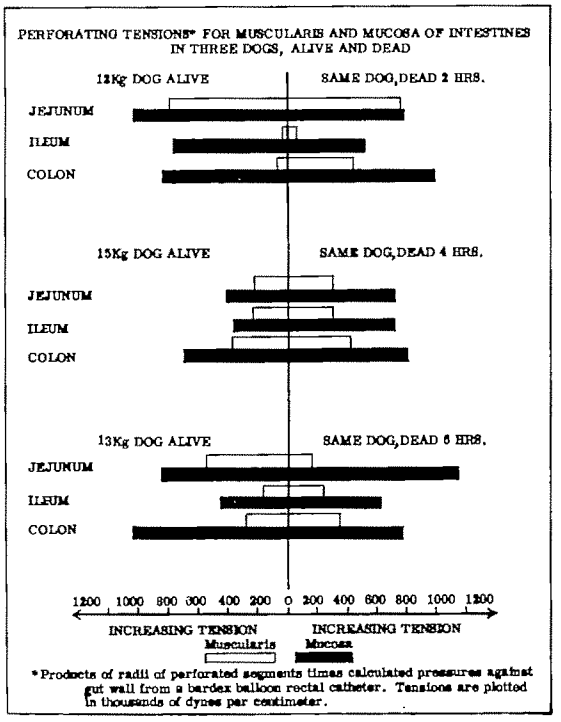


Fig. 3. A diagram showing the perforating tensions for the muscularis and mucosa of intestines in 3 dogs, alive and dead. The perforating tensions are the products of the radii of the perforated segments times the calculated pressures against the gut wall from a Bardex balloon rectal catheter. The tensions are plotted in thousands of dynes per centimeter.

EXPERIMENT NO. 2

DETERMINATION OF HYDROSTATIC PRESSURES FOR BARIUM ENEMA SUSPENSIONS

The measurement of barium enema pressures was approached by measuring the pressures created in a tightly sealed jug when barium suspensions from varied heights were released into a condom in the jug, via $\frac{1}{4}$ or $\frac{3}{8}$ inch internal diameter tubes. Tap water and three different barium suspensions with their surfaces 3, 4, 5, and 6 feet above the mouth of the jug were run into this artificial rectum (Fig. 4). The barium sulfate suspensions used were: 20 per cent U.S.P. barium sulfate, specific gravity 1.15, low viscosity; 20 per cent I.U.M.C. barium,* specific gravity 1.15, medium viscosity; and 40 per cent I.U.M.C. barium, specific gravity 1.30, high viscosity. In these three barium suspensions, the per cent is the number of grams of dry barium sulfate per 100 cc. suspension.

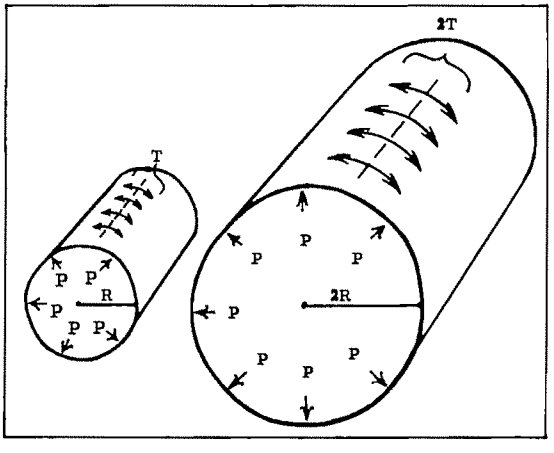


Fig. 2. Diagram of the LaPlace theorem as applied to cylinders. Although the pressure is the same in each cylinder, the tension that is pulling apart the wall of the cylinder with a radius of $2R$ is twice the tension in the cylinder with a radius of R.

* Indiana University Medical Center barium sulfate suspension, Formula No. 319.

TABLE I

HYDROSTATIC PRESSURES FROM BARIUM ENEMA APPARATUS WITH ¼ INCH INTERNAL DIAMETER TUBE: MEASURED PRESSURES* ARE IN CENTER OF EACH BOX; CALCULATED PRESSURES ARE IN LOWER RIGHT HAND CORNER

Height above "rectum" (in feet)	Plastic Tip Wolf "N enema tip" No. 259				Bardex Balloon Catheter #141 (Webber Model)			
	Water	20% U.S.P. Barium sulfate	20% I.U.M.C. Barium	40% I.U.M.C. Barium	Water	20% U.S.P. Barium sulfate	20% I.U.M.C. Barium	40% I.U.M.C. Barium
3	56 / 62	62 / 71	68 / 71	76 / 80	56 / 62	64 / 71	66 / 71	74 / 80
4	80 / 84	88 / 97	96 / 97	108 / 110	80 / 84	88 / 97	92 / 97	106 / 110
5	104 / 107	116 / 123	122 / 123	132 / 139	104 / 107	114 / 123	118 / 123	138 / 139
6	126 / 129	142 / 148	150 / 148	162 / 168	128 / 129	140 / 148	140 / 148	166 / 168

* All pressures are recorded in mm. of mercury; measured pressures are the average of 3 readings multiplied by two.

After an initial bounce of the mercury column with the less viscous materials, the mercury rose to about the same level for a given barium from a given height, no matter what tube or enema tip was used, as expected. Comparison of the measured and calculated hydrostatic pressures in Tables I and II reveals some variation. The variation was even larger initially when the measured pressures were compared with pressures calculated for the distance from the barium level in the container to the center of the jug. Examination of the column being measured revealed that the height above the uppermost level of water in the jug should be used in calculating the hydro-

TABLE II

HYDROSTATIC PRESSURES FROM BARIUM ENEMA APPARATUS WITH ⅜ INCH INTERNAL DIAMETER TUBE: MEASURED PRESSURES* ARE IN CENTER OF EACH BOX; CALCULATED PRESSURES ARE IN LOWER RIGHT HAND CORNER

Height above "rectum" (in feet)	Plastic Tip Wolf "N enema tip" No. 259				Bardex Balloon Catheter #141 (Webber Model)				Miller Metal Tip			
	Water	20% U.S.P. Barium sulfate	20% I.U.M.C. Barium	40% I.U.M.C. Barium	Water	20% U.S.P. Barium sulfate	20% I.U.M.C. Barium	40% I.U.M.C. Barium	Water	20% U.S.P. Barium sulfate	20% I.U.M.C. Barium	40% I.U.M.C. Barium
3	54 / 62	56 / 71	66 / 71	78 / 80	56 / 62	68 / 71	66 / 71	76 / 80	54 / 62	54 / 71	64 / 71	76 / 80
4	78 / 84	84 / 97	94 / 97	104 / 110	80 / 84	96 / 97	92 / 97	104 / 110	76 / 84	86 / 97	90 / 97	108 / 110
5	100 / 108	112 / 123	116 / 123	132 / 139	104 / 107	120 / 123	116 / 123	134 / 139	98 / 107	112 / 123	114 / 123	128 / 139
6	122 / 129	140 / 148	140 / 148	150 / 168	126 / 129	140 / 148	138 / 148	162 / 168	118 / 129	143 / 148	138 / 148	154 / 168

* All pressures are recorded in mm. of mercury; measured pressures are the average of 3 readings multiplied by two.



Fig. 4. The apparatus used to measure hydrostatic pressures: jug filled with water, condom over end of Bardex rectal catheter, tubes, manometer, and barium suspension container. Jug has an internal diameter of 16 cm.

static pressure of this system. In a rigid container filled with a liquid, the pressure is the same everywhere (Pascal's law). A manometer connected to a liquid-filled container will level at the height of the uppermost level of liquid. This level of the manometer becomes the zero mark for measurement of additional pressure, and its equivalent, the uppermost level of liquid in the container, serves as the zero mark for calculation of hydrostatic pressure.

Experiment No. 3

Measurement of Barium Suspension Flow Rates and Calculation of Equivalent Pressures

The flow rates for water and the three barium suspensions of Experiment No. 2 were measured with the enema can at different heights and with several combinations of tubes and enema tips. These flow rates are shown in Tables III and IV. The flow rates in cubic centimeters per second were divided by the effective cross-sectional area of the tube and tip in square centimeters in each case to obtain the average velocity of the barium or water in centimeters per second. The average velocity could not be calculated in most cases because of inability to determine the effective

Table III

Flow Rates and Equivalent Pressures for ¼ Inch Tube: Measured Rates* are in Upper Left Corner; Equivalent Pressures** are in Lower Right Corner

Height of can above table (in feet)	Plastic Tip Wolf "N enema tip" No. 259				Bardex Balloon Catheter #141 (Webber Model)			
	Water	20% U.S.P. Barium sulfate	20% I.U.M.C. Barium	40% I.U.M.C. Barium	Water	20% U.S.P. Barium sulfate	20% I.U.M.C. Barium	40% I.U.M.C. Barium
3	31 (4.6)	29 (4.7)	11 (.67)	1.0 (.006)	27	26	9	0.8
4	37 (6.6)	36 (7.2)	14 (1.1)	1.4 (.012)	32	32	11	1.1
5	40 (7.6)	40 (8.8)	18 (1.8)	1.8 (.020)	36	34	15	1.4

* Flow rates are in cc./sec. through 7 feet of ¼ inch internal diameter tube.
** Equivalent pressures are in mm. of mercury.

TABLE IV

FLOW RATES AND EQUIVALENT PRESSURES FOR ¾ INCH TUBE: MEASURED RATES* ARE IN UPPER LEFT CORNER; EQUIVALENT PRESSURES** ARE IN LOWER RIGHT CORNER

Height of can above table (in feet)	Plastic Tip Wolf "N enema tip" No. 259				Bardex Balloon Catheter #141 (Webber Model)				Miller Metal Tip			
	Water	20% U.S.P. Barium sulfate	20% I.U.M.C. Barium	40% I.U.M.C. Barium	Water	20% U.S.P. Barium sulfate	20% I.U.M.C. Barium	40% I.U.M.C. Barium	Water	20% U.S.P. Barium sulfate	20% I.U.M.C. Barium	40% I.U.M.C. Barium
3	48	46	25	3.6	38	37	16	2.0	80 (4.7)	88 (6.5)	38 (1.2)	4.9 (.024)
4	53	53	32	5.0	46	46	21	2.6	96 (6.7)	96 (7.8)	53 (2.4)	6.7 (.043)
5	60	60	40	6.0	48	50	25	3.4	120 (10.6)	108 (9.8)	69 (4.0)	8.4 (.068)

* Flow rates are in cc./sec. through 7 feet of ¾ inch internal diameter tube.
** Equivalent pressures are in mm. of mercury.

cross-sectional area when tubes and tips of varying internal diameters were used.

For those tube-tip combinations with fairly constant internal diameters, the calculated velocities were converted to equivalent heights of liquid by use of the formula: $V^2 = 2gh$ (Ippen[5]). The equivalent heights of the water or barium suspensions were multiplied by their specific gravities and divided by 13.6 to convert them to a mercury column of equivalent pressure. The equivalent pressures that could be calculated are also listed in Tables III and IV.

EXPERIMENT NO. 4

MEASUREMENT OF RESTING AND SPASM PRESSURES IN COLONS DURING BARIUM ENEMAS

The intrarectal pressures during barium enemas with the bottom of the enema can 4 or 5 feet above the table top were measured in 100 adult patients. A mercury manometer was tapped into the barium enema tube at a short distance "upstream" to the rectum, and the intrarectal pressures were measured after the barium tube was clamped (Fig. 5). This arrangement connected the colon content to the manometer by an essentially nondistensible, liquid-filled tube. It appeared reasonable to assume that the intrarectal pressure was the same as the intracolonic pressure, in the absence of an intervening spastic segment of colon. In fact, Rendleman *et al.*[10] measured rectal and cecal pressures simultaneously in dogs and found the pressures identical below 25 cm. of water; at pressures above 25 cm., the rectal pressures were no more than 10 per cent higher than the cecal pressures.

One might think that measurement of the intrarectal pressure at rest is not a valid measurement for barium enema pressures, but that pressure should be measured with the barium flowing into the rectum. However, if the position of the manometer tap is moved toward the enema can, the manom-

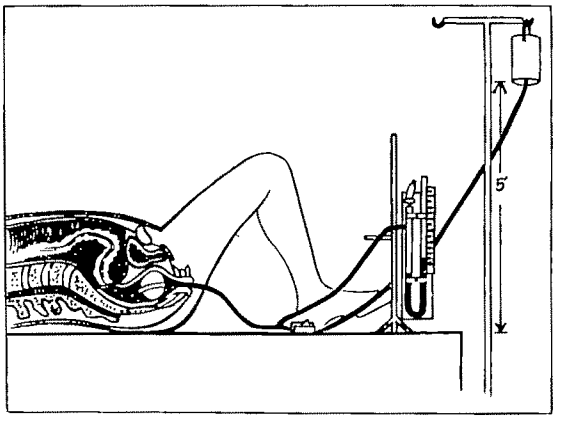


FIG. 5. A diagram of the method used to measure intracolonic pressures. The enema tube is clamped above the manometer tap, and a resting intracolonic pressure of approximately 20 mm. Hg is present.

eter will indicate progressively higher pressures. This gradient of pressure is to be expected, for energy is expended in overcoming the internal friction of the barium and the friction of the tube. The farther the liquid moves down the tube, the less energy is available to be manifested as pressure (Bernoulli's theorem).[5] This variation of pressure with the position of the tap indicates the worthlessness of attempting to measure intrarectal pressure from a tap into the barium tube with the barium flowing.

With the barium tube clamped (Fig. 5), the resting pressure in the colon is fairly steady; quiet respiration does not alter it. As the colon is filled, or as time elapses, the resting pressure may increase or decrease from 2 to 20 mm. Hg, but this change is a gradual one. Straining or spasm increases the intrarectal pressure, but it returns to approximately the same level when the strain or spasm ends. Repeatedly interrupting the barium flow almost always causes the same pressure to appear on the manometer; thus the resting intracolonic pressure is a fairly reproducible finding.

The maximum resting intracolonic pressures were recorded in 83 adult patients with the enema can 4 feet above the table top. These pressures ranged from 8 to 38 mm. Hg, with a mean of approximately 22 mm. Hg (Table v). Table v also shows the confidence limits for anticipated maximum pressures in future cases, calculated according to a method from Fraser and Gutman.[4] Since these pressures were measured in a U-tube manometer, requiring multiplication of the observed readings by two, no odd-valued pressures are included, and accuracy greater than 2 mm. Hg is not possible.

In each of 37 patients, after the maximum resting pressure had been measured for the colon filled with the bottom of the can 4 feet above the tabletop, the barium flow was started again. Then the bottom of the enema can was raised to 5 feet with further filling of the colon; the hose was

TABLE V

STATISTICAL ANALYSIS OF MAXIMUM RESTING PRESSURES IN COLONS WITH ENEMA CAN AT 4 FEET

Pressure (mm. Hg)	Distribution	Confidence limits for maximum pressure to be expected in future cases from same population as sample		
		Confidence Coefficient	$a'_\beta = \sqrt{1 + \frac{1}{n}}\, t_\beta$	Upper Confidence limit $= \bar{x} + a'_\beta s$
8	2			
10	4			
12	4			
14	5			
16	9	.75	.682	26.8
18	6			
20	14	.90	1.300	31.3
22	3			
24	8	.95	1.674	34.1
26	5			
28	8	.99	2.388	39.3
30	5			
32	3	.999	3.214	45.4
34	5			
36	0	.9999	3.922	50.6
38	2			
		.99999	4.562	55.3
n	83			
$\sum x$	1,810	.999999	5.159	59.7
$\sum x^2$	43,884			
$\bar{x}$	21.807	.9999999	5.725	63.8
s^2	53.816			
s	7.3359			

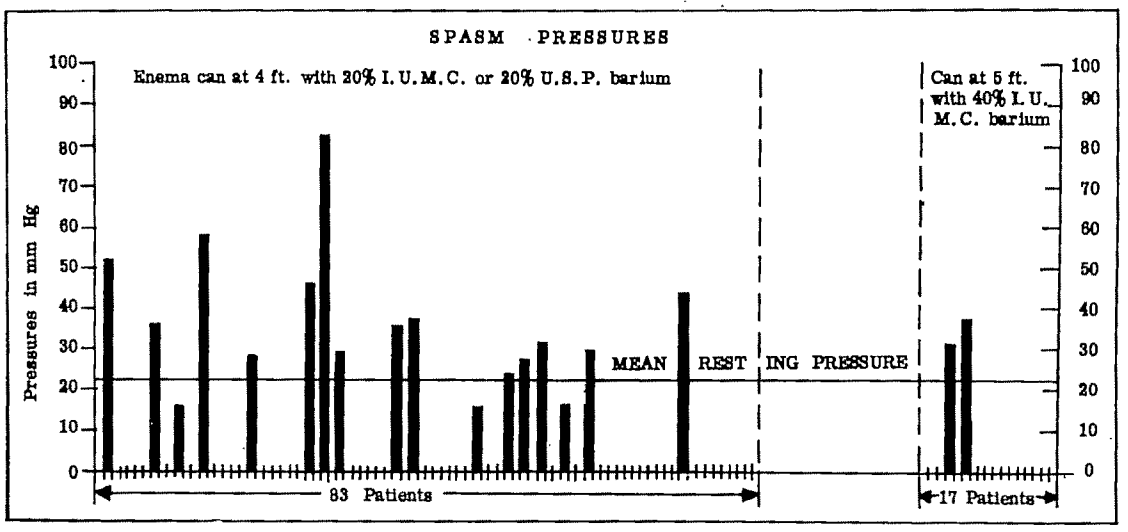


Fig. 6. A diagram of the spasm pressures that occurred in 100 patients during barium enemas; 83 patients received low or medium viscosity bariums; 17 patients received a high viscosity barium from 1 foot higher.

reclamped, and the maximum resting pressure was again measured. A small increase or decrease in maximum resting pressure occurred in some patients, but when the pressures were analyzed as a group, no significant difference was found between maximum resting pressures at 4 feet compared to those at 5 feet.

During these barium enemas, elevations of pressure that ranged from small, transient rises to high pressure cramps persisting for 30 to 60 seconds were encountered. These spasm pressures were measured in the absence of straining; the addition of straining increased the intrarectal pressure an additional 170 mm. Hg in one patient. The spasm pressures observed in 83 patients with the enema can at 4 feet and a barium of low or medium viscosity are shown in Figure 6. The spasm pressures that occurred in 17 patients with the enema can at 5 feet and a high viscosity barium are also plotted in Figure 6. The mean of the maximum resting pressures for filled colons is abbreviated in title and drawn as a line on this figure and the following one. Some of the spasm pressures are lower than this mean value because the maximum resting pressures in some individuals were very low, 8 mm. Hg, for example.

In the 37 patients for whom the enema can was raised to 5 feet after the colon had been filled from a height of 4 feet with a low or moderately viscous barium, new and higher spasm pressures were observed (Fig. 7). A sustained spastic pressure of 112 mm. Hg occurred in 1 patient under these conditions; this pressure was not due to straining, and it was present with the barium tube clamped.

DISCUSSION

In the findings from Experiment No. 1 (Fig. 3) the absence of a definite difference between perforating tensions for dog intestine, whether alive or dead up to 6 hours, validates insofar as possible the application of the Burt[1] data to living human intestine. A portion of Burt's data with pressures converted to mm. Hg is presented in Table VI. These data indicate the pressure tolerance of segments of the colon and ileum. They also demonstrate the effect of diameter on tolerance to pressure (Fig. 2); the small intestine has a greater tolerance for pressure than the large intestine, particularly the cecum or transverse colon, and the colons of infants have a greater pressure tolerance than the corresponding segments of adult colons.

In Experiment No. 2 recognition of the error in calculating pressures from the center of the jug to the barium level in the can points to a potential error in the clinical situation. There is a tendency to calculate the hydrostatic pressure during a barium enema by multiplying the height of the column above the table by its specific gravity. The colon, not the table top, receives the enema. The anus lies about 7 to 9 cm. above the table in most supine patients. Part of the sigmoid colon lies at the level of the anus, but the proximal sigmoid and descending colon are known to lie at a higher level, as diagrammed in Figure 5. This highest level of the colon is estimated to lie about 14 to 18 cm. above the table top. There is usually 16 cm. or more height of barium in the can at the beginning of the enema, and this increment offsets the elevation of the descending colon above the table; one can use the distance between the bottom of the enema can and the table top as an approximation of the total head of pressure. Other variations in the effective height of the enema column occur when the patient is turned, or when spasm occurs in the sigmoid and essentially lowers the uppermost level of barium in the colon. One should be aware of these variables.

If one uses the data in Tables I and II to approximate the hydrostatic pressure between the bottom of the enema can and the table top, 6 to 8 mm. Hg should be added to these values, for both the measured and the calculated values are based on columns that are 8 cm. shorter than 3, 4, 5, or 6 feet.

The data from Experiment No. 3 (Tables III and IV) demonstrate a faster flow rate for water and 20 per cent U.S.P. barium than for 20 per cent I.U.M.C. or 40 per cent I.U.M.C. barium. No suspending agent is present in the first two materials, but the I.U.M.C. bariums contain carboxymethyl-

TABLE VI

BURSTING PRESSURES* OF SECTIONS FROM THE RECTUM,** SIGMOID, TRANSVERSE COLON, CECUM, AND ILEUM (AFTER BURT[1])

Sex	Age	Rectum		Sigmoid		Transverse Colon		Cecum		Ileum	
		Serosa	Mucosa	Serosa	Mucosa	Serosa	Mucosa	Serosa	Mucosa	Serosa	Mucosa
M	5½ mo.	536	646	556	615	318	357	169	307	456	496
F	8 mo.	—	—	278	576	258	332	159	377	—	—
M	11 yr.	—	—	526	605	—	—	—	—	258	397
F	21 yr.	238	258	278	318	139	179	60	139	437	437
F	30 yr.	—	—	119	292	79	214	40	50	179	198
M	30 yr.	—	—	179	188	60	119	—	—	139	139
M	37 yr.	—	—	139	159	—	—	57	99	179	179
M	42 yr.	—	—	198	238	99	149	109	149	139	139
M	42 yr.	—	—	139	139	99	159	—	—	159	318
				119	168						
M	43 yr.	—	—	139	139	89	114	79	119	278	278
M	45 yr.	99	109	139	149	79	89	—	—	278	278
F	50 yr.	—	—	139	159	—	—	—	—	228	258
F	60 yr.	—	—	119	179	40	79	79	99	278	278
F	60 yr.	—	—	134	179	99	119	40	79	—	—
						79	109				
M	62 yr.	—	—	248	258	79	139	60	99	238	238
M	64 yr.	—	—	60	154	89	95	60	60	159	159
M	78 yr.	198	198	119	179	119	149	50	60	248	248

* Segments of gut were perforated with compressed air; rate of pressure increase was about 20 mm. Hg per second. These pressures tabulated in psi in Burt's[1] paper, have been divided by 0.01915 to convert them to mm. Hg.

** Segments of rectum were dissected out of the pelvis, prior to testing.

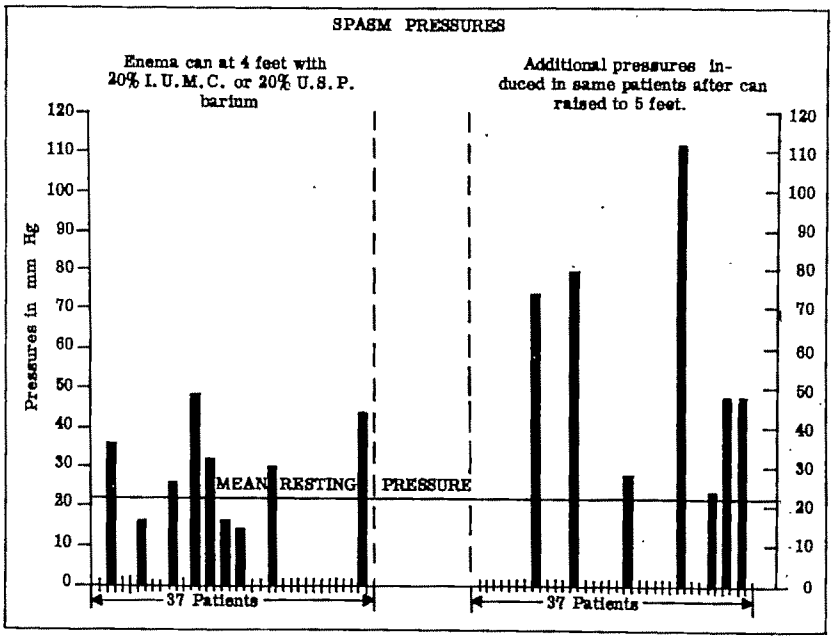


Fig. 7. A diagram of the spasm pressures that occurred in 37 patients who received low or medium viscosity bariums. On the left are the pressures present initially with the enema can at 4 feet; on the right are the new pressures that appeared after the can was raised to 5 feet.

cellulose; there is twice the amount of this suspending agent in the 40 per cent barium as in the 20 per cent barium. The importance of increased viscosity due to the suspending agent is dramatized by comparing the stream of 20 per cent I.U.M.C. barium coming from a 4 foot enema can through a $\frac{1}{4}$ inch tube (Fig. 8*A*) with the dripping ooze of 40 per cent I.U.M.C. barium coming from a 5 foot enema can through a $\frac{3}{8}$ inch tube (Fig. 8*B*). Doubling the amount of suspending agent offsets a wider diameter tube, higher enema can, and higher specific gravity to produce a trickle.

The barium flow rate data of Miller[7] (Table vii) demonstrate the fastest flow rate for U.S.P. barium sulfate in water. The other bariums contain suspending agents and flow more slowly. These flow rates indicate the relative viscosity of a number of barium suspensions.

The equivalent pressures calculated in Experiment No. 3 help to validate the method used in Experiment No. 4; in this experiment pressures were measured in the colon when the barium was not flowing. The difference between resting pressures determined by this method and the true resting pressures is small, for the calculation of equivalent pressures from the kinetic energy of the barium flowing through the various tubes and tips yields pressures ranging from a fraction of 1 mm. to 11 mm. Hg at the most. The increment of pressure to be added to the resting intracolonic pressures measured by the method shown in Figure 5 will almost always be less than 7 mm. Hg. Because of the distribution of the maximum resting intracolonic pressures (Table v), it is doubtful that addition of these equivalent pressures would be recognized except as a trend in a group.

In interpreting the upper confidence limits of Table v, a degree of uncertainty is present because of the size of the sample. For example, in this Table it is estimated that there is only 1 chance in 10,000 of finding a case with maximum resting pressure greater than 50.6 mm. Hg. The 50.6 value is made higher than necessary to compensate for the uncertainty due to the sample size. If we knew the complete

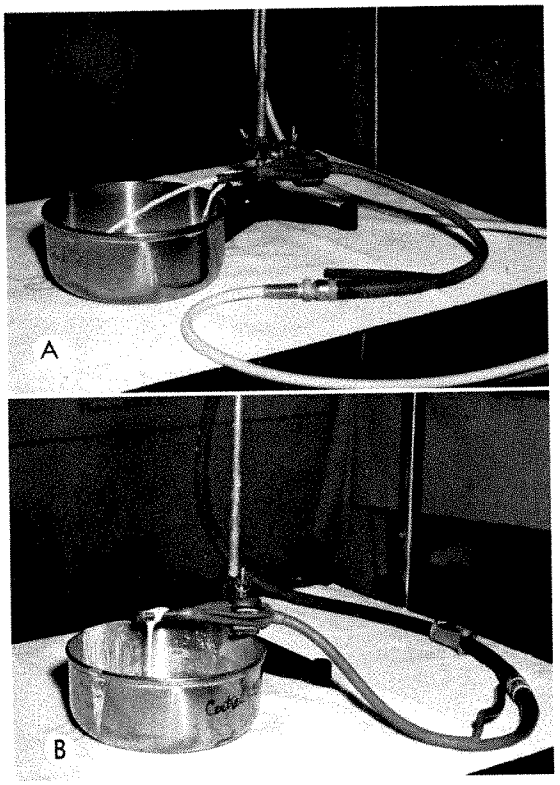


Fig. 8. (*A*) The importance of viscosity. In this photograph 20 per cent I.U.M.C. barium is flowing through a $\frac{1}{4}$ inch internal diameter tube from a can 4 feet above the table. (*B*) In this photograph 40 per cent I.U.M.C. barium is trickling through a $\frac{3}{8}$ inch internal diameter tube with the enema can at 5 feet.

Table VII

Barium sulfate suspension flow rates (after Miller[7]) (for 1 pint of suspension at room temperature through 6 feet of tube. Bottom of enema can is 3 feet above table; 2 quarts of suspension are in the can initially)

Suspensions	Times (in seconds or minutes plus seconds)			
	7/32* inch Tube		3/8 inch Tube	
	20%**	40%	20%	40%
Baridol	1:12	1:48	19	26
Barium Sulfate Compound (Mallinckrodt)	30	33	10	12
Barotrast	1:08	4:44	18	53
Basolac	30	2:09	10	58
Gastropaque	28	36	10	12
I.U.M.C. Barium Suspension	33	5:31	10	1:26
I.X. Barium	28	44	10	14
Micropaque	28	39	10	12
Stabarium, improved	28	37	10	12
Veri-O-Pake	28	38	10	12
Ultrapaque B	1:43	11:07	25	2:07
U.S.P. Barium Sulfate in distilled water	13	20	8	10

* Most 1/4 inch internal diameter enema tubes are really 7/32 inch internal diameter.

** % concentration is by weight of barium sulfate per 100 cc. suspension in I.U.M.C. barium suspension or U.S.P. barium sulfate in water. Concentration of other suspensions is by weight of commercially available dry powder per 100 cc. suspension.

population distribution, it is likely that fewer than 1 in 10,000 would be greater than 50.6 mm. Hg.

It must also be said that the method used to measure these resting pressures does not rule out small pressure elevations due to slight, undetected contractions of the colon. No tracing of the pressures was made.

It is believed that the spasm pressures observed during these enemas are due to a slowing or stopping of the barium column by the Type II waves or Type IV waves of the colon described by Code *et al.*[3] When movement of the barium column slows or stops, energy formerly used to overcome friction or impart velocity to the column is manifested as pressure (Bernoulli's theorem). The highest pressures are probably due to Type IV waves (mass peristalsis) stopping the barium column. With the anal canal blocked by a balloon, these high pressures often persisted for 30 to 60 seconds, until the spasm subsided.

Comparison of the spasm pressures observed in Experiment No. 4 with the hydrostatic pressures for the same conditions (Tables I and II) reveals that the spasm pressures with the can at 4 or 5 feet are

always less than the hydrostatic pressures for the same conditions. Several authors have reported reflux into the enema tube with the can 23 to 26 inches above the table[8] or 1½ to 3 feet above the table;[11] some of these patients strained during these observations. If an enema can is placed with its bottom 3 feet above the table top (60–86 mm. Hg total head of pressure) and straining is discouraged, it is believed that Type IV waves alone will rarely develop enough contractile force to overcome the total head of pressure, reflux barium into the tube, and contaminate it with feces.

When examining the colon with a viscous barium in approximately 40 per cent concentration, one can elevate the bottom of the barium can safely to a height 5 feet above the table top in order to speed the filling of the colon. Although a potential pressure head of approximately 130 mm. Hg is present, the high viscosity of these suspensions causes so sluggish a flow that when spasm occurs, the spasm pressures are not only relatively low (Fig. 6), but they are also slow in onset. A 20 mm. Hg pressure rise requires about 20 seconds to develop fully. Subjectively, these enemas with a high viscosity barium from an enema can at 5 feet are as comfortable as an enema can be.

In the sample of 17 colons filled with a high viscosity barium (Fig. 6), the incidence of spasm appeared less also. When an enema can filled with a low viscosity barium was elevated (Fig. 7), the flow rate was increased, and not only were higher spasm pressures observed, but also new spasm pressures appeared in previously resting colons. This incidence of spasm varying directly with the speed of filling suggests a receptor mechanism in the colon wall that is sensitive to the rate of dilatation of the colon and that initiates contraction of a segment of the colon.

What are the pressures on the side of the colon proximal to the spastic segment? They cannot be measured by the method shown in Figure 5 unless the patient has a proximal colostomy. Use of an ingested transducer in a telemetry capsule could answer this question. At this point it can be stated that observations during Type IV waves reveal the same amount or less barium in the ascending colon after a Type IV wave than was present before the contraction. In no patient in over 100 enemas did a previously unfilled terminal ileum fill during a Type IV wave. These observations are consistent with the general concept that a Type IV wave moves colonic content distally. It is believed that proximal intracolonic pressures are usually the same or less than intrarectal pressures during a barium enema.

Giving enemas with a relatively low viscosity barium suspension from 4 or 5 feet routinely is not advocated. The incidence and height of spasm pressures increase with the height of the can, and the associated cramps are painful. And, although it is not possible to set an over-all pressure tolerance for the colon because of the variation in segmental tolerance (Table VI), the presence or absence of a disease such as ulcerative colitis, the varying competency of the ileo-cecal valve,[10] and the presence or absence of a balloon catheter obstructing the anal canal, it appears that repeated high spasm pressures in a large group of patients would cause perforation.

Because the resting pressure in the colon is low, mean maximum resting pressure being about 22 mm. Hg, it may be possible to develop an enema tip with a valve mechanism that would eliminate most cramps due to spasm pressures.

Satisfactory pressure studies during air contrast enemas could be done by combining cineroentgenography with a constantly recording pressure transducer in the rectum or in an ingested telemetry capsule. This method would probably give more information about the nature of colonic contractions and could also check and extend the findings of Experiment No. 4.

SUMMARY

1. High viscosity barium suspensions can be given safely and comfortably with the

bottom of the enema can 5 feet above the table top (essentially at the ceiling) because of the marked loss of pressure head with high viscosity liquids.

2. When intestine perforates, it obeys the law of LaPlace, *i.e.*, the tearing apart of intestinal wall is not due to pressure alone, but is due to the product of the change in pressure across the wall times the radius of the intestine.

3. The measurements of bursting pressures and radii obtained on autopsy specimens can be applied to living human intestine.

4. The mean value of the maximum resting pressures in colons is about 22 mm. Hg.

5. A sustained spasm pressure of 112 mm. Hg, not due to straining, was produced in one patient during a low viscosity barium enema from 5 feet.

6. When low viscosity barium suspensions are given from 4 or 5 foot heights, many spasm pressures are seen. The spasm pressures are always less than the available hydrostatic pressure. The incidence and height of these spasm pressures increase with elevation of the enema can.

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TRANSMISSION OF POLIO VIRUS VACCINE BY CONTAMINATED BARIUM ENEMA WITH RESULTANT ANTIBODY RISE*

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FECAL contents frequently contaminate the barium enema tubing and can. Contamination with feces takes place even when the enema can is several feet above the patient. This has been conclusively proven by pressure and bacterial studies by one of us,[5] by radioactive isotope and bacteriologic studies of Steinbach *et al.*[9] and by observations of Nathan,[7] Pochaczevsky *et al.*[8] and Dryfuss *et al.*[1]

An analysis of the methods by which barium enema examinations are performed shows that complete asepsis is lacking. Usually the enema tip is disposable; however, the enema set (can, glass, connecting rods, tubing, and tip) is rarely autoclaved, or disposed of, after each examination.

It is the purpose of the authors to call attention to the fact that transmission of polio virus vaccine by contaminated barium enemas with resultant antibody rise does occur and that possible cross infection can be prevented by autoclaving or by using a plastic disposable enema kit.

METHOD AND RESULTS

A separate sealed disposable plastic barium enema system* prepackaged with 10 ounces of micronized barium was used on each individual patient. The disposable enema system was filled in a retrograde fashion without opening the bag, through the long tubing with 2 quarts of water. Two cc. of orimune,† a live, oral monovalent Sabin strain Type II polio virus vaccine was injected into each plastic bag. Acute blood samples were drawn from 7 patients before the enema procedure. The patients were screened for history of polio virus or contact with the disease before and during the study. The same 7 patients were given a routine barium enema using different separate disposable barium enema systems as described; each containing the regular Sabin intraoral dose of polio virus vaccine. Convalescent blood samples were drawn from each patient about 3 weeks later. Serum neutralization antibody titers were done on both the acute and convalescent blood serum. Table I shows that 6 out of 7 patients had a 2 to 32 fold rise in antibody titer within 3 weeks. One patient had no rise. Two patients had a 2 fold rise, 2 patients had a 4 fold rise, 1 patient had a 16 fold rise and 1 patient had a 32 fold rise.

TABLE I

Patient	Acute Serum	Convalescent Serum	Rise
	(9-20-62) 9-21-62	(10-11-62) 10-12-62	
L. C.	1:8	1:8	None
L. S.	1:32	1:64	2 fold
A. P.	1:64	1:128	2 fold
M. B.	1:8	1:32	4 fold
J. G.	1:32	1:128	4 fold
L. R.	1:8	1:128	16 fold
J. D.	1:8	1:256	32 fold

DISCUSSION

In this group of patients, no precautions were taken to prevent contamination of the patient's hand (and thus possible secondary contamination of their gastrointestinal tract). However, we are dealing with the

* Manufactured by EZ-Em Co., Inc., 130 Maple Street, Great Neck, Long Island, New York.
† Supplied by Lederle Laboratories.

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normal every day procedure by which barium enemas are given and this is of prime importance. There is tremendous variability in the time and quantity of polio virus excreted from an immunized or infected patient.[2,3,6] It seems reasonable to assume that at sometime during the excretion phase of an individual patient, the quantity of polio virus in the colon and terminal ileum would be equal to the routine Sabin dose used in this study.

Excretion may contain causative organisms of disease which primarily affect systems other than the gastrointestinal tract such as ova, parasites, hepatitis virus, mycobacteria and coxsackie virus.

It is obvious that sterilization of enema equipment or use of disposable apparatus (a check valve would be contaminated on one surface and could be thoroughly contaminated by filterable viruses) would solve the contamination problem. Over 13 million fluoroscopic procedures[4] are done annually in the United States, a substantial number employing barium enemas. With the use of enemas (both barium and water), the possibility of contamination exists and may cause an epidemiologic route for diseases with long incubation periods.

SUMMARY AND CONCLUSIONS

The results of the reported study show that a virus, such as the attenuated polio virus, can be transmitted via a contaminated barium enema. The polio virus vaccine was used as an indicator or sample virus and it is concluded that the transmission of polio virus implies transmission capabilities of other viruses and bacteria from contaminated barium or water enemas.

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ESOPHAGEAL DIVERTICULUM AS CAUSE FOR UNSUCCESSFUL CHOLECYSTOGRAPHY*

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PREHEPATIC diversion of ingested contrast medium into an esophageal diverticulum is a potential, albeit rare, cause for nonvisualization of a normal gallbladder during cholecystography. Only one such case has been recorded in recent years. Among approximately 21,000 cholecystographic examinations at the Ochsner Clinic during the past 10 years, the case to be reported is the only known instance of trapped opaque medium in a diverticulum resulting in nonvisualization of the gallbladder.

REPORT OF A CASE

On February 10, 1960, a 77 year old, white, machine shop owner was examined at the Ochsner Clinic because of epigastric discomfort, lower thoracic pain accentuated by swallowing, and increasing regurgitation of mucoid fluid and food particles for several months before admission. He had noticed increasing anorexia, loss of 12 pounds in weight, and occasionally streaks of blood in the regurgitated material.

The gallbladder was not visualized in the cholecystogram made on February 11, 1960. In the largest roentgenogram obtained, contrast medium was observed in the lower portion of the thorax (Fig. 1), and a diagnosis of trapping of medium in an esophageal diverticulum was suggested. On the next day, a gastrointestinal roentgenologic study proved the presence of a saccular, pulsion diverticulum, 8 cm. in diameter with a mouth 3.5 cm. wide, arising from the right posterolateral aspect of the esophagus, 3 inches above the diaphragm (Fig. 2)

The diverticulum was resected by Dr. Alton Ochsner on February 26, 1960. Recovery was uneventful, and the patient was discharged from Ochsner Foundation Hospital on March 8, 1960. A cholecystographic examination, on March 15, 1961, revealed a normal gallbladder which concentrated the medium well and contained no gallstones (Fig. 3).



Fig. 1. Large roentgenogram during cholecystography shows opaque medium in lower thoracic area.

DISCUSSION

Cholecystography is one of the most reliable and useful of roentgenologic procedures. A successful examination requires oral ingestion of an opaque medium, its absorption from the small intestine, its excretion by the liver, and its passage from the biliary ducts into the gallbladder, where it is concentrated by absorption of water by the mucosa.

Failure at any stage of this series of events may result in failure of visualization of the gallbladder. The commoner

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causes of prehepatic diversion of ingested cholecystographic medium are vomiting of the substance before it passes out of the stomach, sufficient obstruction at the gastric outlet to prevent an adequate amount of substance from reaching the intestine, and extreme intestinal hypermotility, which transports the medium through the intestine before it can be absorbed. Trapping of medium in a diverticulum of the upper gastrointestinal tract is a potential, but rare, cause for failure of absorption of the ingested medium. In the case reported by Lake,[1] the diverticulum arose from the upper part of the eophagus. May and Moussard[2] recorded an instance of trapping of the medium in a duodenal diverticulum. No report of trapping in a gastric diverticulum has been discovered. In the case herein reported, the true nature of the disease was recognized because one roentgenogram revealed enough surrounding anatomic structures to permit incidental visualization of the opaque medium in the lower portion of the thorax. Trapping of the substance in an esophageal diverticulum was then suspected. Examination of the esophagus by barium study showed a large diverticulum. After diverticulectomy, a normal gallbladder was visualized cholecystographically.

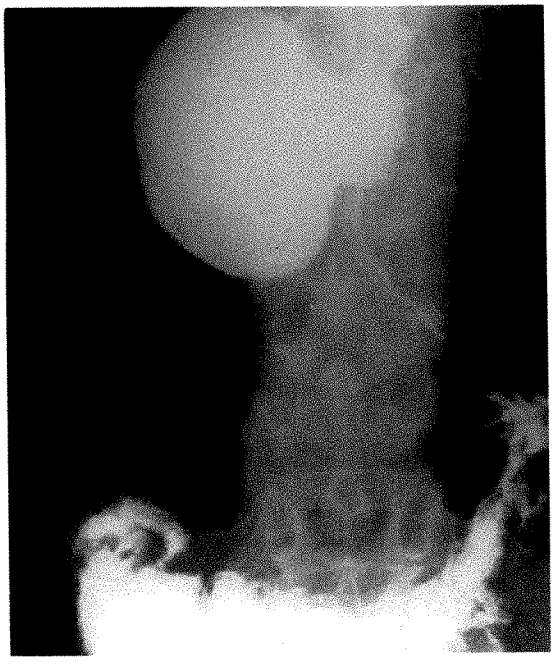


Fig. 2. Esophagram proves presence of large lower esophageal diverticulum.



Fig. 3. Cholecystogram shows normal visualization of gallbladder after resection of esophageal diverticulum.

If on the usual roentgenograms, the gallbladder is not visualized, it would seem advisable to obtain at least one large roentgenogram. This will help eliminate such conditions as unusual location of the gallbladder, retention of medium in the stomach, or trapping of medium in a diverticulum of the upper gastrointestinal tract.

SUMMARY

A case in which a large, lower esophageal diverticulum trapped the opaque medium which was ingested for cholecystography is described. Reports of such unusual prehepatic diversion of the contrast substance are rare, only one other having been discovered in a brief review of medical reports. If the gallbladder is not visualized on roentgenologic study, possible causes other than cholecystic disease must be considered. If at least one large roentgenogram is obtained, errors due to abnormal position of the gallbladder and abnormal pre-intestinal retention of medium may be largely eliminated. In the case reported, a

normal result of cholecystographic study was obtained after resection of the esophageal diverticulum.

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DEMONSTRATION OF A ROUND WORM IN THE COMMON BILE DUCT

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THERE are several reports in surgical journals concerning biliary ascariasis; however, a review of the literature shows a paucity of articles dealing with the roentgenologic observations. The object of this communication is to familiarize the reader with the roentgenographic appearance of a round worm in the common bile duct, which was visualized on an intravenous cholangiogram of the reported patient.

REPORT OF A CASE

A 28 year old married woman was admitted to the hospital on May 20, 1963 with severe intermittent colicky pain in the right upper abdomen of 8 days' duration. She frequently vomited during the attacks. She had had a similar attack about 5 years previously, which lasted for a period of 5 to 6 days. On examination, she was found to be poorly built and obviously dehydrated. Abdominal palpation showed right upper quadrant tenderness but no mass. She was not jaundiced. Intravenous baralgan and intramuscular pethidine relieved the symptoms for ½ to 2 hours only and massive doses of analgesics had to be repeated.

A clinical diagnosis of acute pancreatitis was made. Total white blood cell count was 10,400 with 70 per cent polymorphonuclears, 20 per cent lymphocytes, 8 per cent eosinophils and 2 per cent monocytes. Serum amylase was 146 units (normal 70–200 units).

A plain roentgenogram of the abdomen did not demonstrate any abnormality. Oral cholecystography was not performed because of persistent vomiting; however, intravenous cholangiography was carried out. Cholangiograms showed a smooth linear filling defect in the entire length of the common bile duct, which was of a normal caliber (Fig. 1 and 2). The gallbladder showed normal concentration of contrast medium and no intrinsic abnormality was demonstrated. The contrast material flowed to the duodenum normally (Fig. 2).

Because of the abnormality in the common bile duct, an exploratory operation was carried out on the next day and an adult living worm 15 cm. in length was removed. In the postoperative period, antihelminthic treatment was instituted and a number of round worms were noted to pass in the stools.

DISCUSSION

Intestinal ascariasis is fairly common in India, as is its frequent demonstration on barium meal studies. In our 40 year experience in this clinic, where gallbladder investigations are frequently carried out, we have not before encountered a single case of biliary ascariasis. We have contacted several colleagues in Delhi and in other centers in Northern India and have not been able to trace any other such case. In



FIG. 1. One-half hour cholangiogram. A smooth linear filling defect is seen in the entire length of the common bile duct. The duct is not dilated.

a recent publication Wright, Dorrough and Ditmore[6] reported an incidence of 1.5 per cent of surgically proved biliary ascariasis in hospital patients in Korea. Wang *et al.*[4] gave an analysis of 141 surgically proved cases from Tsingfad, China. In these reports there is no mention of diagnosis being made by roentgenologic studies. The only recent case in which a round worm was demonstrated on intravenous cholangiograms and laminagrams is reported by Cywes and Krige[1] from South Africa.

Although we are aware of this condition, it was considered only as one of the differential diagnoses along with air in the common bile duct or numerous small translucent calculi filling it. In retrospect, however, the appearance seems quite characteristic and perhaps once seen, should not be missed again.

SUMMARY

A case is reported of a round worm in the common bile duct visualized on intravenous cholangiograms. It is felt that a definitive diagnosis of the condition can be made from the roentgenographic findings.

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We wish to thank the surgeon, Dr. T. J. Aggarwal of the City Clinic for allowing us to publish this case. We are grateful, also, to Prof. S. Chawla for her encouragement and help.



Fig. 2. Two and one-half hour cholangiogram. There is normal filling of the gallbladder. Contrast medium is seen in the duodenum. The filling defect is again demonstrated.

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Vol. 91, No. 4

OPERATIVE CHOLANGIOGRAPHY*

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THE most important objective of operative and postoperative cholangiography is to visualize defects within the biliary duct system, usually indicating the presence of stones. The reliability and the accuracy of cholangiography have been thoroughly reviewed and discussed. Unfortunately, many of these examinations are performed under adverse conditions with less than optimal roentgenographic results. Under such circumstances the diagnostic accuracy of the procedure is diminished. Some surgeons have considered the incidence of diagnostic error from operative cholangiograms to be sufficiently high to warrant the elimination of the examination and proceed with the surgical exploration of the extrahepatic biliary ducts in spite of the increased mortality or morbidity which might result from this extended procedure. However, even surgery has its limitations in the evaluation of the bile ducts and their contents. Recently, the introduction of endoscopy has improved the surgical evaluation of the bile ducts at the time of operation.

It was our purpose to determine the reliability of operative cholangiography by *in vivo* and *in vitro* studies of the bile ducts. The following factors have been evaluated: type and concentration of the contrast medium, size of the common duct, size of the defect within the common duct, the grid ratio, kilovoltage, milliamperage, and exposure time.

The *in vivo* studies were performed on consecutive patients by postoperative T-tube cholangiography, using standard factors (kilovoltage range 70–80, milliamperage 200–400, exposure time 0.1–0.2 seconds, 8:1 grid and target film distance 36 inches) with three contrast media—iodopyracet (diodrast) 35 per cent, methylglucamine diatrizoate (renografin) 30 per cent, and sodium iodipamide (cholografin) 20 per cent.

The common duct size was measured directly on the roentgenograms and all cholangiograms were graded as excellent, good, fair, poor, or unsatisfactory. The cholangiograms were studied for diagnostic quality rather than for the subjective impression obtained upon viewing the roentgenogram. Great importance was placed on good and uniform filling of the bile ducts with good contrast and adequate penetration of the opaque column.

The *in vitro* studies were performed as a separate project. Lucite blocks, measuring 18×18×2.5 cm., were used to simulate the bile ducts. Holes of 5, 10, 15, and 20 mm. diameter were drilled in each block to duplicate the common duct. Roentgenograms of the lucite block with contrast medium in the holes were made with and without a masonite phantom (20×20×20 cm.). Subsequent studies were all done with a phantom. Two types of grids were used with ratios of 8:1 and 16:1. Gallstones were selected and placed within the drilled holes. Inasmuch as the stones were not of uniform size, subsequent studies were then carried out using artificial stones made of a dental plastic material. Under such circumstances, it became possible to make stones of equivalent size and as small as 2 mm.

By varying the roentgenographic factors it was feasible to evaluate those which were significant in affecting the diagnostic quality of the cholangiogram. Taken into consideration were: (1) Size of the patient; (2) kilovoltage range; (3) grid ratio; (4) milliamperage; (5) time of exposure; (6) target film distance; (7) size of roentgen-ray tube focal spot; (8) contrast medium and concentration; (9) lack of motion of the pa-

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tient; (10) size of bile duct; (11) size of stone; (12) size of stone in relation to the size of duct; (13) use of compression; (14) cineroentgenography; (15) type of film used; (16) processing of roentgenograms; and (17) use of collimator or cone.

A total of 81 *in vivo* postoperative T-tube cholangiograms was available for evaluation. In a number of instances, serial cholangiograms of the same patient but with the use of different contrast media could be studied. Inasmuch as the size of the common duct is an important factor in determining the quality of the examination, these cholangiograms have been compared according to the common duct size (Table I). It was found that when the common duct was 11 mm. or larger all of the three contrast media produced cholangiograms of good or excellent diagnostic quality. However, with a common duct size of less than 11 mm., renografin 30 per cent and diodrast 35 per cent yielded better results than cholografin 20 per cent and of these two media, the latter was more effective when the duct size was less than 5 mm.

The factors and some of the results obtained in the *in vitro* studies are given in Table II.

The phantom studies showed that an optimal set of factors for detecting a 3 mm. stone in a 15 mm. duct was 95 kv., 200 ma., 1 second with a 16:1 grid. However, *in vivo* examination would require the use of a much shorter exposure time and perhaps a higher milliamperage.

It was difficult to determine the critical size of a stone which could be detected in the common duct in the *in vitro* experiments. It was apparent that stones of at least 2 or 3 mm. in size are readily demonstrated if the common duct is not larger than 5 mm. in diameter and probably detectable in common ducts up to 10 mm. As the duct size increases, there is a greater likelihood that a stone of such small size would be obscured by the surrounding volume and density of the contrast medium. It is then that other factors already mentioned may begin to play important roles in increasing or decreasing the probability of demonstrating the defect within the duct.

TABLE I

PERCENTAGE CONSIDERED EXCELLENT OR GOOD IN A TOTAL OF 81 T-TUBE CHOLANGIOGRAMS WITH 3 DIFFERENT CONTRAST MEDIA

Common Duct in mm.	20 per cent Cholografin	30 per cent Renografin	35 per cent Diodrast
Less than 5	0	66	100
6 to 10	71	100	100
11 to 15	100	100	100
16 to 25	100	100	100

DISCUSSION

Several authors have studied the physical factors which contribute to the roentgenographic demonstration of gallstones. Cocchi[2] and Wegelius *et al.*[6] advocated the use of a high kilovoltage technique; Hornykiewytsch and Stender[3] recommended a kilovoltage range of 60 to 65 with an exposure of 120 to 200 ma. seconds. Samuel[4] expressed concern over the use of a high density opaque medium which might obscure small stones.

Virtama[5] recently completed an interesting study of these factors and found that it was possible to detect a 1 mm. stone at 50 and 60 kv. when the opaque medium concentration was 5.0 gm. per 100 ml. of water. This stone is about one-half the size of those visible at 100 kv. in the same concentration. Virtama observed no differences with the various types of contrast media used, but noted that the detectability of the stones was impaired by a low concentration of contrast medium and by a high kilovoltage. He also pointed out that a decrease in the size of the tube focal spot from 1 mm. to 0.3 mm. by decreasing the geometric blur improved the chances of detecting calculi. Roentgenographic contrast according to Virtama is increased when a small field size (9×12 cm.) is used. It is also increased by the use of low voltage and a high concentration of iodine. High voltage

Table II

CHOLANGIOGRAPHY FACTORS AND RESULTS

Variable	Result
Contrast media Renografin 30% Diodrast 35% Cholografin 20%	Diodrast 35% was most opaque
Kilovoltage 60 to 110 kv.	The kilovoltage with a phantom must be at least 70. No improvement with 110 kv.
Milliamperage 200 to 400 ma.	200 ma. was better
Time 0.1 to 1 second	For *in vivo* work, should be 0.1 or less
Grid Ratios 8:1 and 16:1	Slightly better with 16:1
Phantom—with and without	All details superior without the phantom
Target film distance constant 36 inches	Shorter target film distance can be used for patients with a thicker abdomen in order to keep the exposure time short
Stones 2–10 mm.	Smaller stones were undetected in ducts larger than 15 mm.
Size of the field (effective cones, collimation)	Roentgenographic detail improved with proper cones and collimation
Thickness of the patient	Decreased roentgenographic quality with increase in the size of the patient
Size of ducts (5, 10, 15, 20 mm.)	Stones usually readily detected in the smaller ducts may not be detected in the larger ducts

has been of value in the study of the intestinal tract, especially the colon, where the technique has been applied for detection of polypoid lesions. However, in the gastrointestinal tract barium sulfate is ordinarily employed as the contrast medium; because of the increased penetrability of the roentgen rays due to different absorption characteristics, the iodine containing compounds do not offer the same advantage in the biliary tract. Virtama also points out that very high milliamperage-second values are not practical since the long exposure time results in increased blurring (due to motion of the patient) and loading of the roentgenographic tube. In his experience small stones were found to be obscured by underexposure in the case of a very dense (opaque) biliary tract.

In a study by Ashmore *et al.*[1] the effects of various concentrations of opaque media were evaluated. An 8 mm. stone could always be detected when neo-iopax (25 to 50 per cent) and diodrast (17.5 to 35 per cent) were used. A 2 mm. stone was not detected with neo-iopax 50 per cent and was seen in one-fifth of the neo-iopax 25 per cent studies. These authors concluded that small stones in an undilated duct were readily seen; however, they emphasized that a negative or normal cholangiogram should be accepted with some misgivings.

Another approach at improving the quality of the T-tube cholangiogram has been the use of air contrast with the introduction of gas following the contrast medium. This technique has been infrequently used and thus cannot be properly evaluated. It might be anticipated that such a study would permit the detection of smaller stones than those generally visualized on the routine cholangiogram, but the technique is difficult and the results may prove inconsistent.

In recent years cineroentgenography has also been tried for the examination of the biliary tract; however, the roentgenographic image quality is inferior to that obtained with conventional roentgenography. Although some advantages may be seen due to a more dynamic technique, these are more than offset by the decrease in the roentgenographic quality. Improvements in cineroentgenography and television amplification may lead to greater

utilization of this method in the future exploration of the biliary duct.

From our studies and a review of the literature, it appears that factors which influence roentgenographic quality, in general, are also applicable to T-tube cholangiography. The exposure time should be short enough to avoid blurring caused by respiration or movement. This would usually be 0.1 second or less. Target film distance may be reduced to accommodate the heavier patient, and compression should be applied with greater frequency in all patients, and most certainly in the obese group. Collimation of the roentgen beam or use of a cone is highly desirable and effective. If other factors can be maintained at their proper level, some advantage may be obtained with a 16:1 grid. Opaque media of "intermediate density" usually yield better visualization of stones. Good roentgenographic technique will generally suffice when the common duct is less than 10 mm. in caliber; however, when the common duct is larger, it is necessary to vary the factors so that small stones may not be obscured.

CONCLUSION

Various factors contributing to the roentgen detection of bile duct calculi have been analyzed by *in vivo* and *in vitro* studies. Three different contrast media have been evaluated for use in operative cholangiography and it was found that opaque media of "intermediate density" gave the best diagnostic results.

Careful selection of factors to be used and precise roentgenologic techniques should do much to increase the diagnostic accuracy of operative cholangiography and, thereby, decrease the postoperative complications of biliary tract surgery.

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CONGENITAL ABSENCE OF THE INTRAHEPATIC BILE DUCTS*

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CONGENITAL absence or atresia of the intrahepatic bile ducts is a rare anomaly, first described in 1865. At the time of the last review, in 1958, 10 acceptable cases had been collected and a subsequent case was reported in 1961.[1–11] Two of the previously described cases have been from this institution.[1] The roentgenographic aspect of the condition as noted in these 2 cases as well as in 7 others is the basis of this discussion.

In the previous reports, it has been suggested that the disease is the result of a congenital absence of the intrahepatic bile ducts. However, bile ducts have been found to be present in the early weeks of life, and they have remained for variable periods of time. We feel that the cause is most likely an intrinsic defect in the liver that produces bile stasis at the preductal level and, either from atrophy or direct damage to bile duct epithelium, results in progressive loss of intrahepatic bile ducts. When the condition is progressive, a unique clinical and roentgenographic pattern develops. Serial histologic studies, or autopsy, demonstrate disappearance of intrahepatic bile ducts. We feel that absence, rather than

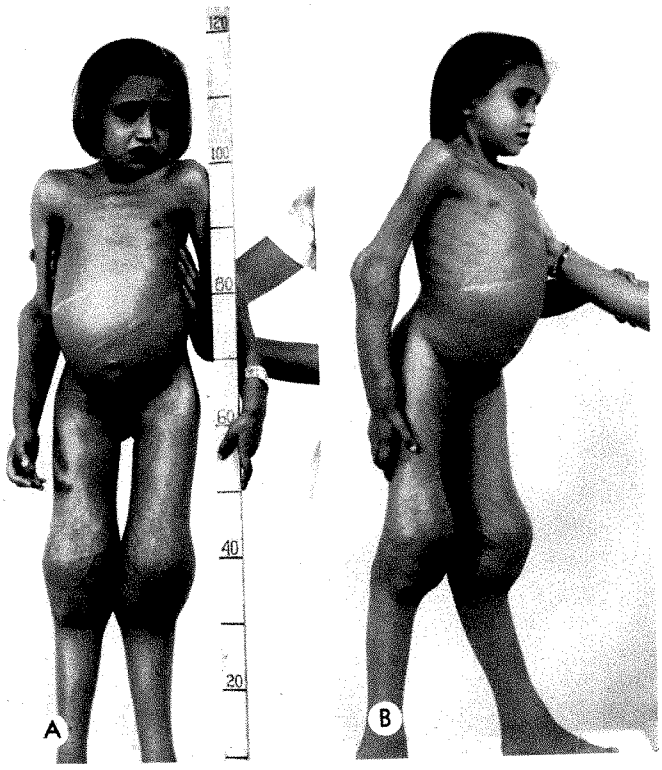


FIG. 1. (*A* and *B*) Photographs of our oldest living patient shown with marked swelling over the bursae, a protuberant abdomen and generalized debility.

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Fig. 2. Photograph showing the typical lesions of xanthomatosis that these patients develop.

atresia, is a more appropriate term for this rare and perplexing condition.[6]

CLINICAL FINDINGS

Since the detailed clinical aspects of these cases are being published elsewhere,[6] we will only summarize the clinical picture (Fig. 1, *A* and *B*). The disease begins with a progressive obstructive jaundice, with its onset between 2 days and 2 months of age, although the infant remains comparatively well. A pronounced elevation of the serum lipids occurs, which after a variable period of time, usually a few months, is followed by cutaneous xanthomata (Fig. 2) and severe pruritus. Subsequently, the lipid level may fall and the xanthomata may recede while the pruritus remains. The defect responsible for the preductal bile stasis may well be associated with lipid metabolism. Some patients with a mild form of the disease do not show progressive jaundice, have only a minimal evidence of liver disease and develop neither the clinical nor roentgenographic changes.

In patients with the progressive form of the disease, liver function is preserved for long intervals. These children live much longer than the larger group of patients with extrahepatic bile duct atresia, a condition which more closely adheres to the definition of atresia. The major clinical manifestations in our group of 9 patients were severe icterus in 6, xanthomatosis in 7, and cholesterol levels in excess of 700 mg. in all 9. Hypersplenism, portal hypertension and anemia developed as relatively late complications in the older children. Several of the patients showed easy bleeding as well.

The liver at operation or autopsy is large, smooth, soft and green, quite unlike the small, shrunken and extensively scarred livers seen in extrahepatic biliary atresia.

Histologic examination of the biopsy specimens in 7 of our cases initially showed bile pigment in the liver cells and canaliculi, large pale liver cells with two or more nuclei, occasional multinucleated giant cells and occasional necrosis of an individual cell with focal polymorphonuclear infiltration. The bile ducts were small and rarely contained bile (Fig. 3). Serial biopsies showed a progressive loss of bile ducts with only a mild diffuse fibrosis; the fibrosis present in the later phase is much less severe than that seen in extrahepatic biliary obstruction and tends to preserve the lobular pattern (Fig. 4). The degree of loss of the bile ducts varied from individual to individual and also varied in different parts of the liver at postmortem examination. The progressive loss of bile ducts and the variability seen clinically and microscopically are inconsistent with an atresia and suggest an intrinsic liver defect.

Bone marrow aspirations, done late in the course of the disease, have demonstrated both the presence of fat laden histiocytes and hyperplasia of erythroid elements. It is therefore tempting to consider the possibility that some of the roentgen changes are similar to those seen in a storage disorder. These foam cells have also been seen in the spleen and kidneys.[9] The number of such cells in the marrow has not been high, but the bone marrow aspirations have been performed when the bone changes were already advanced and in some cases after the blood lipid levels have decreased.

We became aware of the possible specificity of the roentgenographic changes in the skeleton when the advanced changes observed in 1 of our patients were duplicated in another. A review of our 9 cases re-

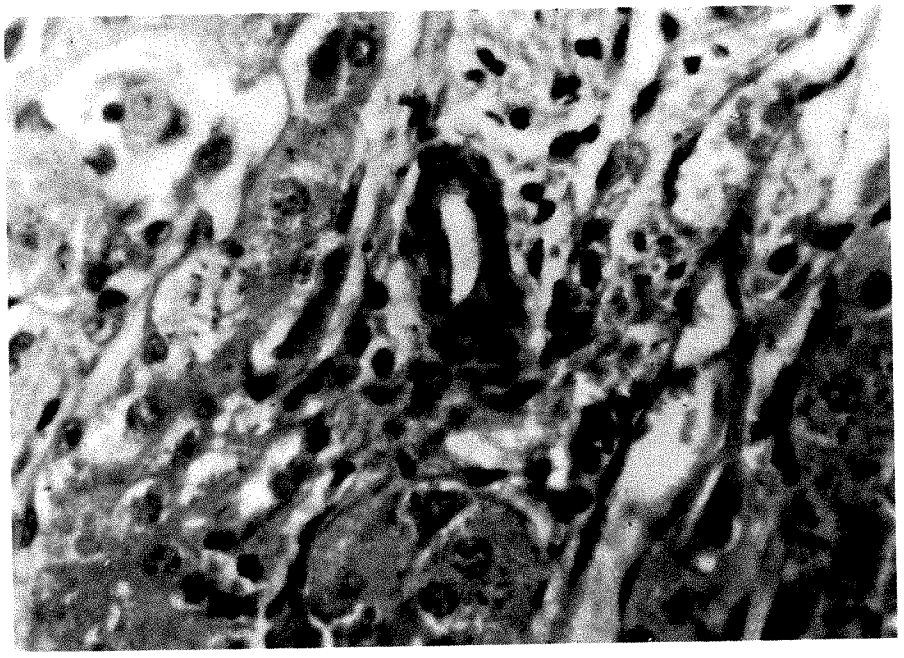


Fig. 3. Photomicrograph of a portal area from an early biopsy showing pale multinucleated liver cells and a small empty bile duct.

vealed that these skeletal findings were present in the entire group in varying degrees and that the progression occurred in a predictable pattern dependent on the degree of bile duct deficiency.

ROENTGEN FINDINGS

Roentgenographically, the skeleton is normal at birth (Fig. 5). As the child develops, stress lines appear in the metaphyseal areas and the skeleton becomes

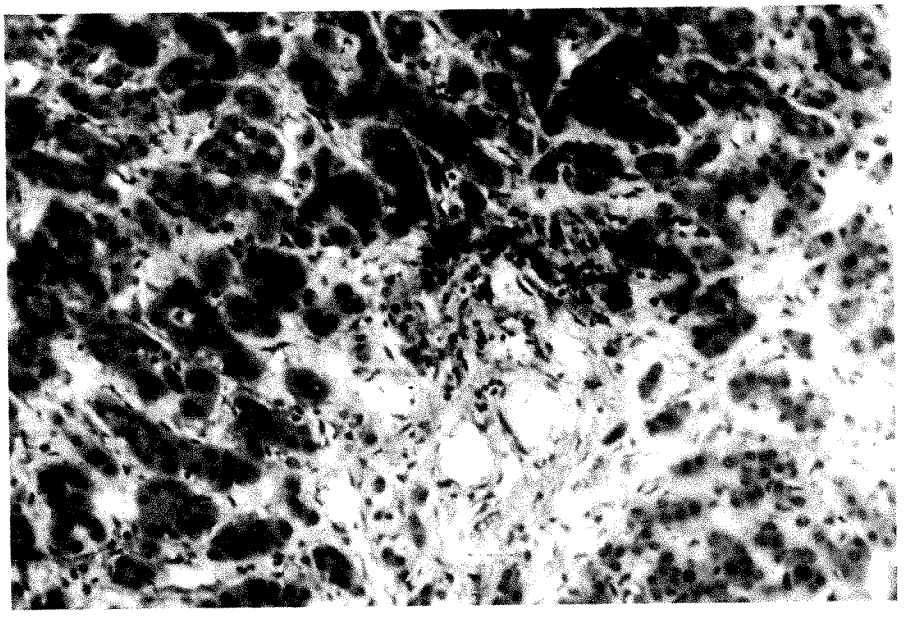


Fig. 4. Photomicrograph at autopsy shows a portal area with diffuse mild fibrosis and preservation of the lobular pattern.

progressively demineralized. These changes are probably not a manifestation of protein deficiency such as have been described in other types of liver disease,[11] for these patients do not show a diminution of serum protein or a reversal of the albumin-globulin ratio. The generalized debility associated with their illness may well contribute substantially to the lack of mineral content. Demineralization is associated with a progressive and severe failure of funnelization of the metaphyses, the exact cause of which is not clear. As the disease progresses, the diaphyseal portion of the bone, which had a relative elongated and thinned appearance due to the failure of funnelization,

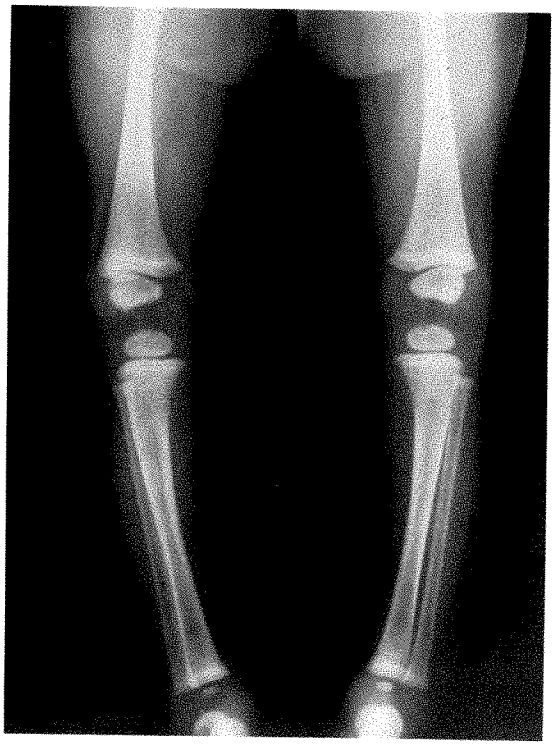


Fig. 5. Roentgenogram of the lower extremities at 5 months of age shows normal appearance save for prominent "stress" lines. (Figures 5 through 15, inclusive, are of the same patient.)



Fig. 6. Roentgenogram at 7 years of age shows the demineralization with resorption of primary trabeculae and failure of funnelization of the metaphysis. Swelling over the right knee is apparent.



Fig. 7. Lateral roentgenogram of the right knee made at the same time as Figure 6.



Fig. 8. Overpenetrated frontal roentgenogram showing the knees as they appear currently.

shows a marked increase in width, a very thinned cortex and a prominent secondary trabecular pattern which results in thick, clubbed and poorly mineralized bone (Fig. 6; and 7). These changes in the skeleton are generalized and are most marked in areas of maximum growth (Fig. 8; and 9, *A* and *B*). The upper and lower extremities are involved, with the lower extremities showing the most pronounced changes (Fig. 10; 11; 12, *A* and *B;* and 13). The exception is the calvarium, which remains normal, quite unlike the response more commonly seen in children with marrow hyperplasia.

During the course of the disease, these children develop bleeding into, or swelling of, the bursal sacs of the extremities (Fig. 14, *A* and *B;* and 15). One of our children first developed symptoms in the right knee following immobilization for a fracture. Subsequently, she had swelling in both

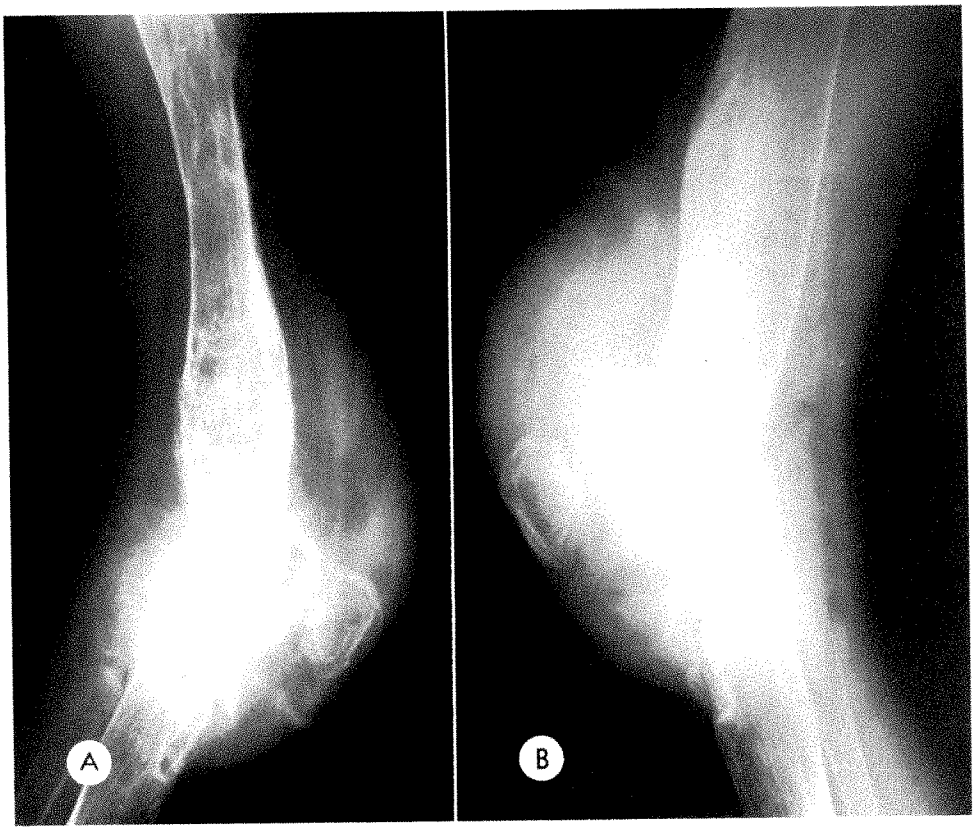


Fig. 9. (*A* and *B*) Lateral roentgenograms of the knees as they appear currently show the marked osseous and soft tissue changes.

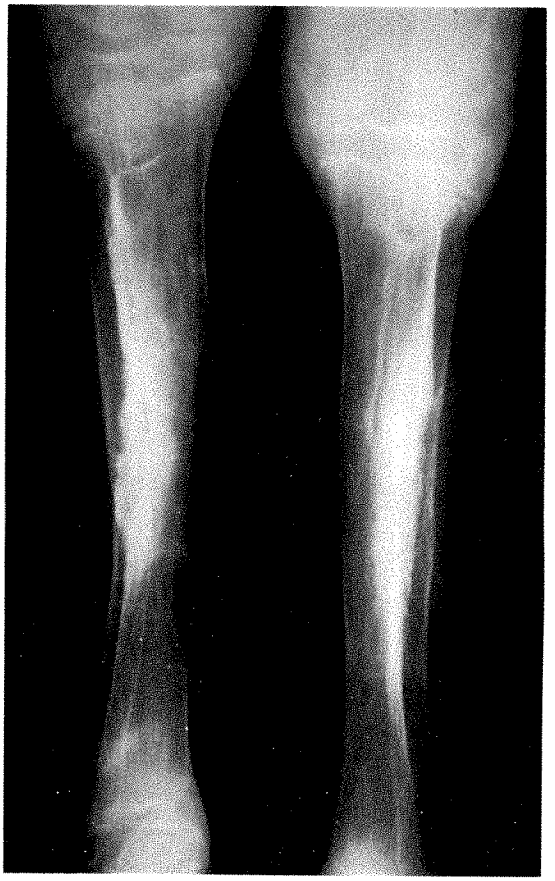


Fig. 11. Roentgenogram of the forelegs as they appear currently showing widening of the diaphysis and cortical hyperostosis, probably associated with bleeding.

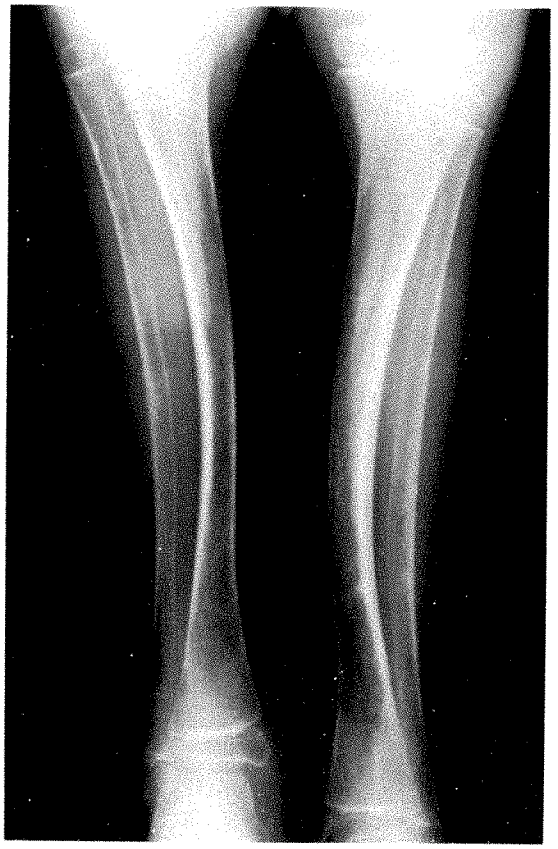


Fig. 10. Roentgenogram of the forelegs in 1957 at the age of 7 years showing demineralization and apparent elongation, secondary to metaphyseal deformity.

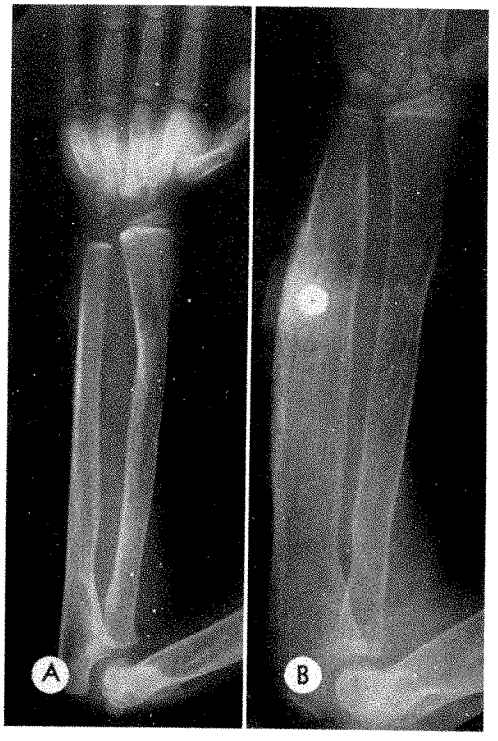


Fig. 12. (*A*) Roentgenogram of the arm and (*B*) one made 5 years later show the same changes as noted in the lower extremities.



Fig. 13. Roentgenogram of the hands as they appear currently shows severe involvement with widening of the metacarpals and phalanges.

knees, both ankles, wrists, bursae about the elbows and in the temporomandibular area. The swelling about the knees was originally felt to represent a hemarthrosis and, indeed, in this patient some of the early bleeding may have been into the joint. Subsequently, the swellings of the bursae have been most noticeable about the knees

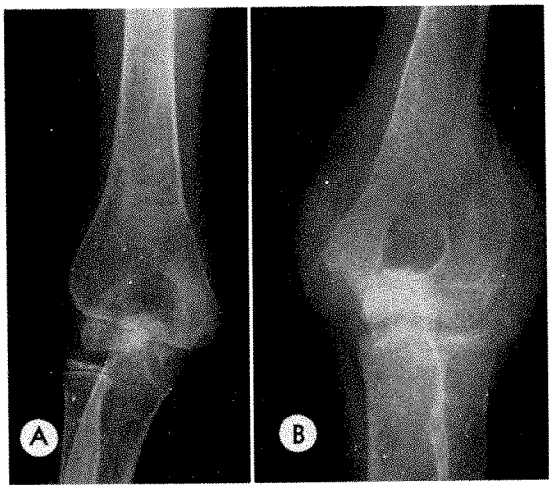


Fig. 14. (*A* and *B*) Roentgenograms showing prominent swelling over the elbow joint and prominent osseous changes, both of which have been progressive.



Fig. 15. Lateral roentgenogram of the foot and ankle showing the same changes as noted in the knees and elbows.

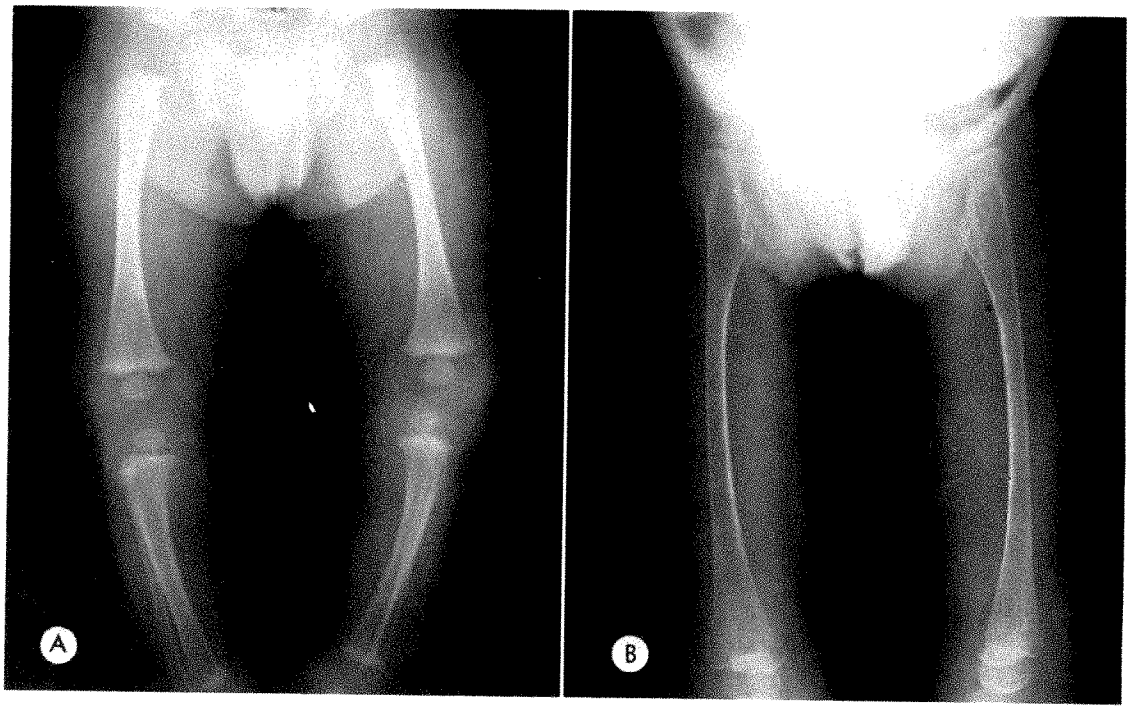


Fig. 16. (*A* and *B*) Roentgenograms of the patients whose skeletons were normal at birth then showed progressive demineralization and apparent elongation from the metaphyseal failure of funnelization.

and have been prominent in 3 of our 9 patients and present in 2 others (Fig. 16, *A* and *B;* 17; 18, *A* and *B;* and 19). Limitation of joint motion in our group was present in only 1 individual, and involved only one knee. The patient with the most severe roentgenographic changes of the extremities developed cortical hyperostosis along the shaft of the long bones, which may well be related to subperiosteal bleeding. Fracture with minimal trauma has been prominent late in the disease. One of our patients has had 12 fractures in 8 years; healing of these fractures has been uneventful. Another patient, early in the course of her illness, showed typical vitamin deficiency rickets which responded readily to therapy and did not recur. Interestingly enough, the roentgen changes which subsequently developed in this patient were a combination of the features of healed rickets and of absence of intrahepatic bile ducts (Fig. 20; and 21). Rickets is frequently seen in patients with extrahepatic biliary atresia during the period of rapid growth, which is usually in the last half of the first year.

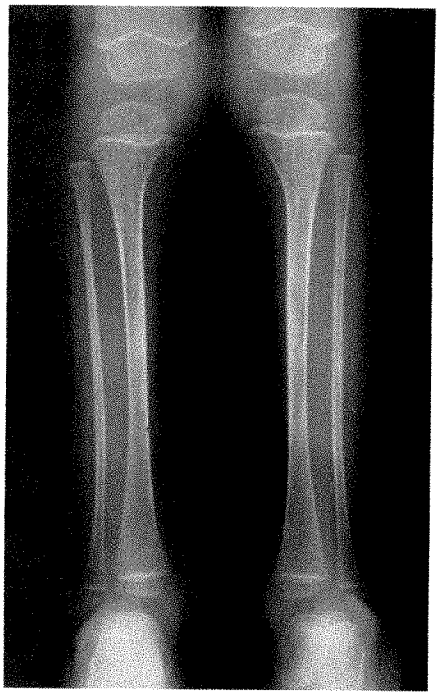


Fig. 17. Roentgenogram of the lower extremities of the same patient shown in Figure 16. The configuration of the medial tibial metaphysis was seen in several of the patients as the metaphyseal deformity developed.

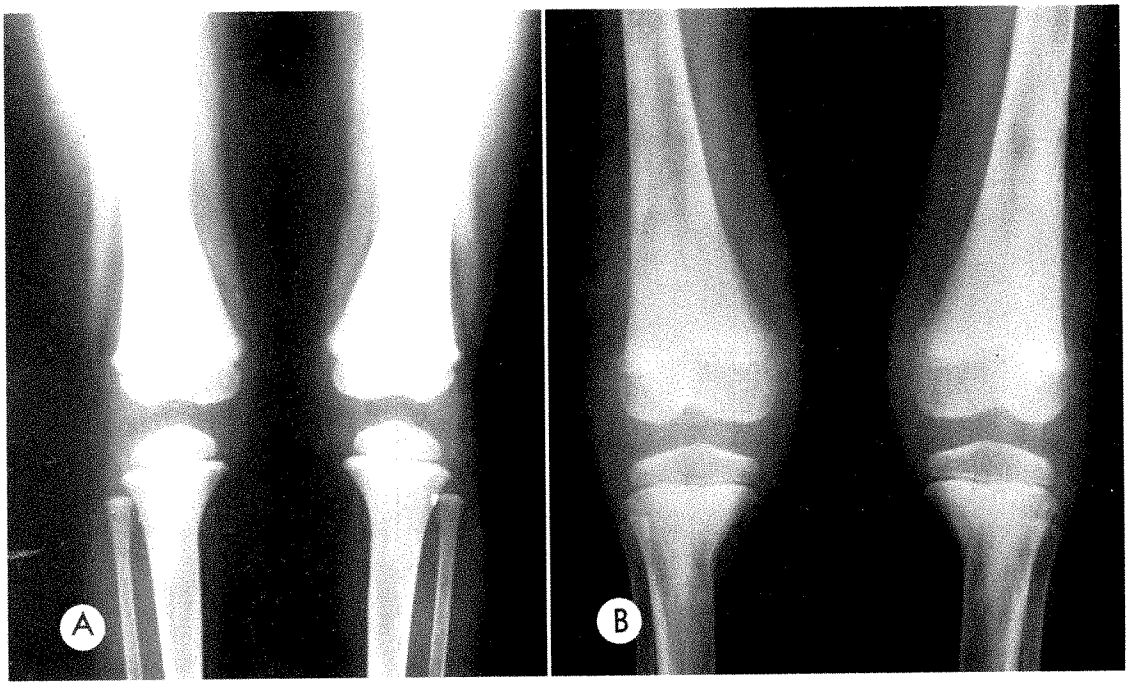


FIG. 18. (*A*) Roentgenogram of the knees and (*B*) one made 3 years later show the development of typical changes in the metaphysis and soft tissues.



FIG. 19. Lateral roentgenogram of the knee of the patient shown in Figure 16.

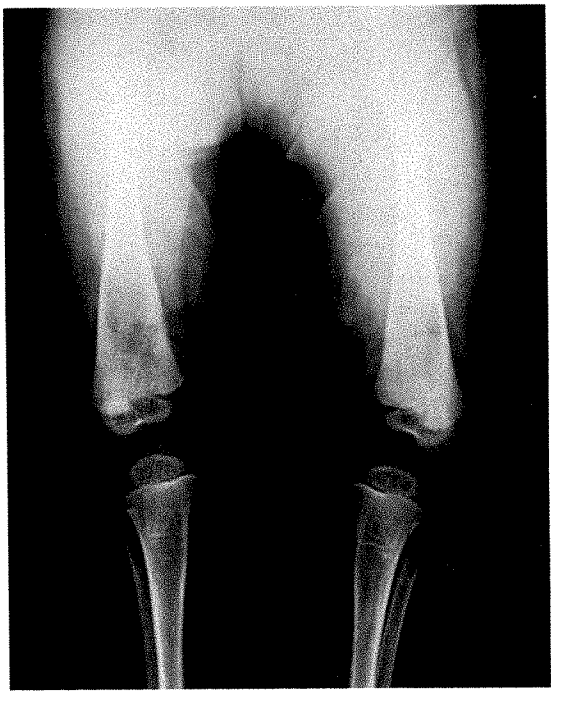


FIG. 20. Roentgenogram of the knees late in the disease of a patient who developed rickets shows the metaphyseal failure of funnelization and the changes of healed rickets.

While the children with absent intrahepatic bile ducts retain a state of relative well being, they do not show periods of rapid growth; both growth and maturation are slow. Esophageal varices have been seen in slightly less than one-half of our patients in association with hypersplenism and portal hypertension; these develop as a late complication of the disease.

While the findings noted above are unlike those of any other storage disease, the failure of normal metaphyseal shape and resorption of primary trabeculae suggest this type of disease process more so than any other osseous manifestation of systemic disease.

SUMMARY

Absence of the intrahepatic bile ducts is a rare anomaly probably due to an intrinsic defect within the liver which, when progressive, develops a unique clinical and roentgenographic pattern. The clinical picture is one of progressive obstructive jaundice, hypercholesterolemia, usually with xanthomatosis, slow gradual liver deterioration with hypersplenism, portal hypertension and, late in the disease, a tendency to bleed easily.

The skeleton, normal at birth, becomes progressively demineralized and shows progressive lack of funnelization and cylinderization, accompanied by progressive swelling of the bursae on the extensor surfaces of the extremities. The pattern and sequence of these changes has been consistent and relative to the degree of hepatic involvement. We have not seen this sequence in any other condition.

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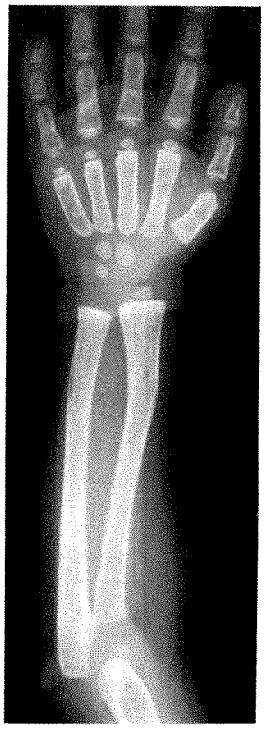


Fig. 21. Roentgenogram of the forearm and hand of the patient shown in Figure 20 demonstrates the same type of change in this area.

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ROENTGENOGRAPHIC, ANGIOCARDIOGRAPHIC AND PATHOLOGIC FINDINGS IN CONGENITAL CARDIAC DISEASE ASSOCIATED WITH AGENESIS OF THE SPLEEN*

REPORT OF A CASE

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AGENESIS of the spleen is almost universally associated with multiple and complex congenital anomalies of the heart.[1,3] These anomalies frequently include defects of the atrial and ventricular septa, transposition of the great vessels, severe pulmonary stenosis or atresia and anomalies of veins, both pulmonary and systemic. Abnormalities in the position of the heart are common. Partial or total situs inversus may be present. The spleen is absent, and the left lobe of the liver is enlarged.

The present case represents a particularly good example of the roentgenographic and angiocardiographic features of one type of cardiac malformation that may occur in the "asplenic syndrome."

REPORT OF A CASE

A female infant, 5 weeks of age, was admitted to the University of Minnesota Hospitals with a history of cyanosis since birth. No murmurs were present.

Clinical Findings. Electrocardiographic study showed frequent premature ventricular contractions, normal axis, and signs of marked right atrial enlargement and right ventricular hypertrophy. Very little left ventricular activity was evident.

In a smear of the peripheral blood, Howell-Jolly bodies were identified.

After completion of the diagnostic studies, including roentgenography and angiocardiography (to be described), a diagnosis of asplenia with congenital cardiac disease was made. A surgical attempt to relieve the obstruction to pulmonary flow was unsuccessful and the infant succumbed.

Roentgenologic Findings. Roentgenograms of the thorax (Fig. 1, *a* and *b*) showed that the size of the heart was within normal limits. The apex of the cardiac density appeared somewhat triangular. The soft tissue density in the superior mediastinum showed a very slight convexity of the lateral margin, bilaterally. This was a little more obvious on the right side and suggested the presence of an enlarged right aortic arch. In addition, there was a homogeneous opacification of the left upper abdominal quadrant indicating enlargement of the left lobe of the liver. This contributed to a horizontal orientation of the lower margin of the liver. The barium swallow study showed the stomach to be on the right side. A forward angiocardiogram was made so that the anatomic features of the cardiac malformations might be demonstrated.

During the angiocardiographic study, films were exposed at the rate of 5 per second. Initial opacification of the inferior vena cava indicated that this structure was malpositioned and lay to the left of the midline. Large veins draining the enlarged left lobe of the liver and a left superior vena cava were opacified by reflux flow (Fig. 2, *a–d*).

Angiocardiograms made immediately subsequent to this defined an enlarged common atrium well filled with contrast material (Fig. 3, *a–d*). In addition, an enlarged, triangular ventricular chamber filled. Also, at this stage, slight filling of an enlarged aorta was noted. No contrast material was detected in the pulmonary trunk or its branches.

Subsequent angiocardiograms showed complete filling of the enlarged and malpositioned aorta. The aorta arose along the left upper border of the heart and arched to the right of the thoracic spine. The aortic arch passed over the right main stem bronchus, and the descending

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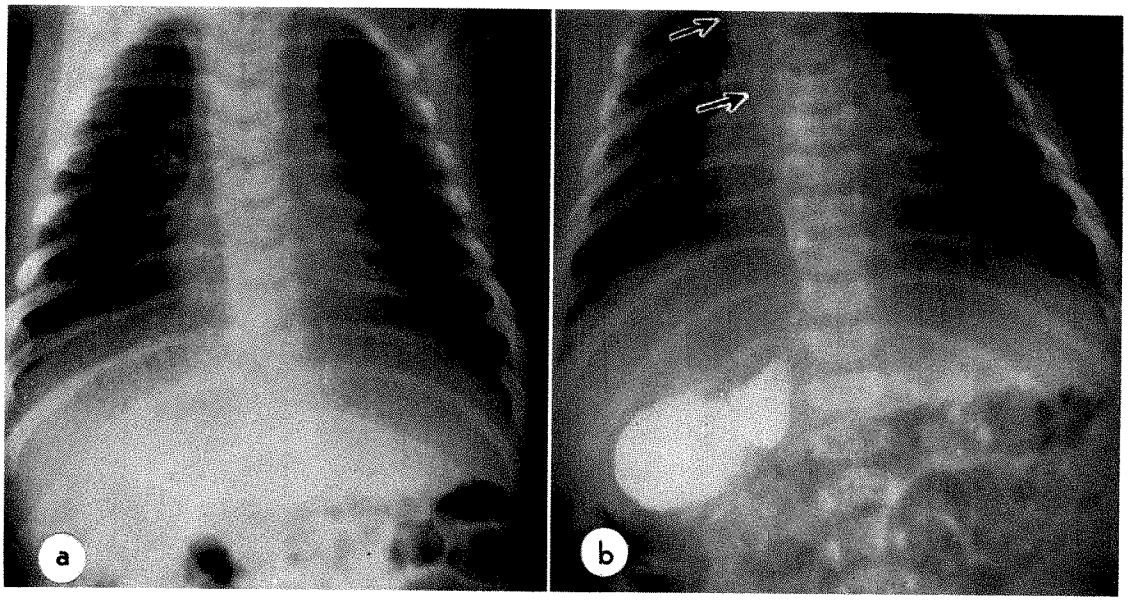


Fig. 1. (*a*) Frontal roentgenogram of the thorax. Cardiac size not remarkable. The configuration of the heart is unusual in 2 respects. The apex of the heart is somewhat triangular, and there is a convex soft tissue prominence in the superior mediastinum on the right side representing the enlarged aorta. The pulmonary vasculature is slightly decreased. Horizontal orientation of lower edge of liver. (*b*) Barium study shows that the stomach is on the right side. The soft tissue density of the enlarged right-sided aorta (arrows) is better seen here than in *a*.

aorta, except for its lowermost part, was on the right side. Following opacification of the aortic arch, there was opacification of the pulmonary trunk and central pulmonary vessels, all of which were narrow. It was noted that the pulmonary vasculature filled by way of a ductus-like structure which appeared to arise either from the aortic arch or from one of the branches of the aortic arch. This vessel descended vertically to the left of the thoracic spine to communicate with the left pulmonary artery. The forward angiocardiogram was interpreted as showing a common atrium, a ventricular septal defect, transposition of the great vessels in the position of corrected transposition and severe obstruction to pulmonary arterial flow.

Pathologic Findings. Necropsy revealed asplenia, a right-sided stomach, malrotation of the mesentery, a nearly symmetrical liver and complex cardiovascular malformations (Fig. 4).

Transposition of the great vessels was evident, with the aorta, which was wide, arising anteriorly and to the left of the comparatively narrow pulmonary trunk. The aortic arch passed over the right main bronchus. The branches from before backward were a left-sided innominate artery (from which in turn the left common carotid and subclavian arteries arose), the right common carotid artery and the right subclavian artery. A patent ductus arteriosus extended between the proximal part of the left subclavian artery and left pulmonary artery.

The interior of the heart exhibited a thin strand of tissue running near the midline between the anterior and posterior walls of the atrial portion of the heart. The picture was like that usually called "common atrium." Bilateral superior venae cavae were present, each joining its respective side of the common atrium. The inferior vena cava was on the left side and after receiving the left hepatic vein terminated in the left side of the common atrium. The right hepatic vein pierced the diaphragm and joined the right side of the common atrium at the anticipated location of the inferior vena caval orifice. No coronary sinus was present.

The atrioventricular mechanism was represented by a single valve which led from the common atrium above into a common ventricle below.

At the anterior basal aspect of the common

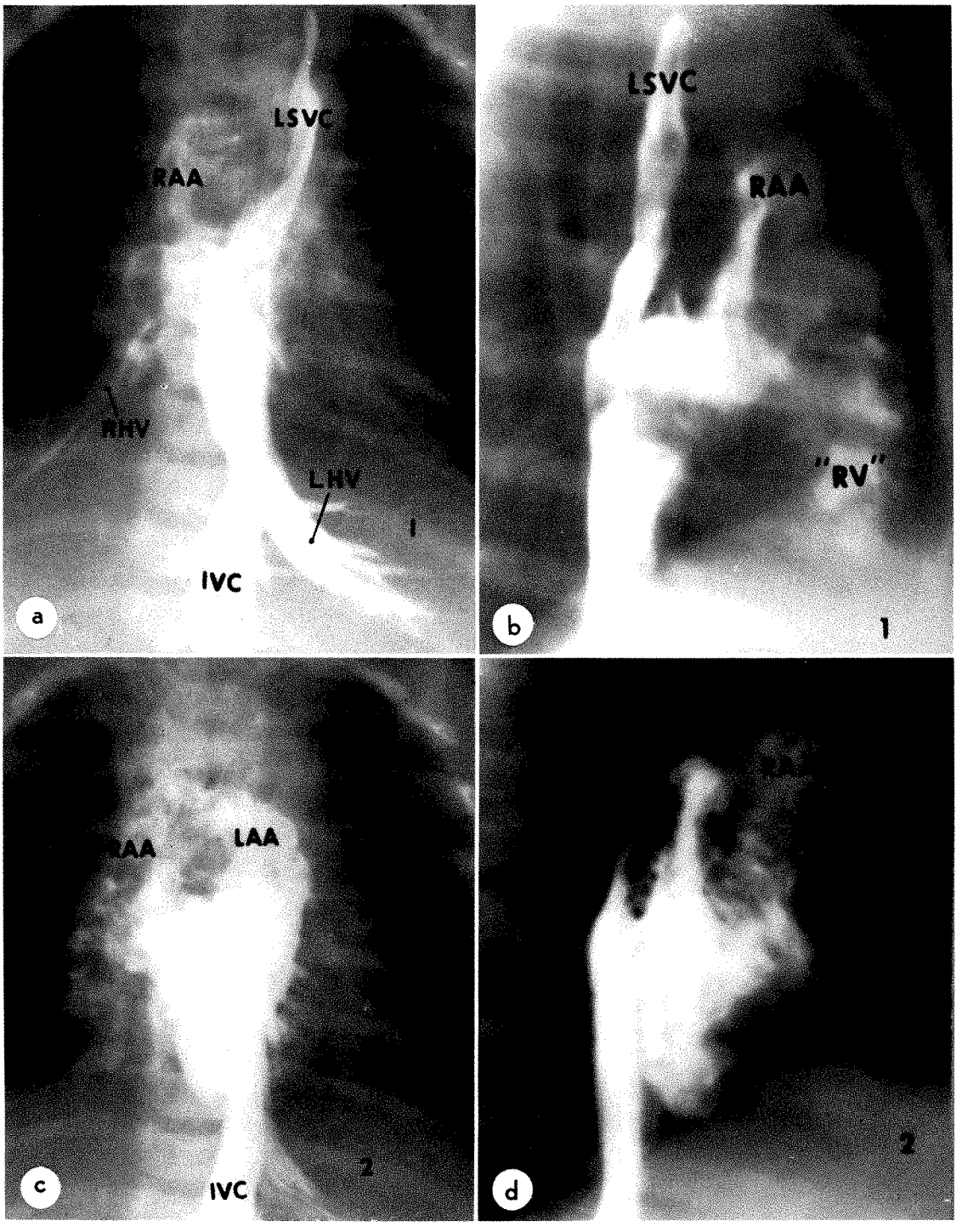


FIG. 2. Forward angiocardiograms. Films were exposed at the rate of 5 per second. Films are numbered according to their position in the study. (*a* and *b*) Frontal and lateral views, respectively. Contrast material in left-sided inferior vena cava (IVC) and in left hepatic (LHV) and right hepatic (RHV) veins. Simultaneous filling of an atrial chamber, right atrial appendage (RAA) and left superior vena cava (LSVC). In the lateral view a small amount of contrast material is seen in the common ventricle ("RV") which at the time of the procedure could not be distinguished from a right ventricle. (*c* and *d*) Frontal and lateral views, respectively. The left (LAA) and the right (RAA) atrial appendages are outlined with contrast material.

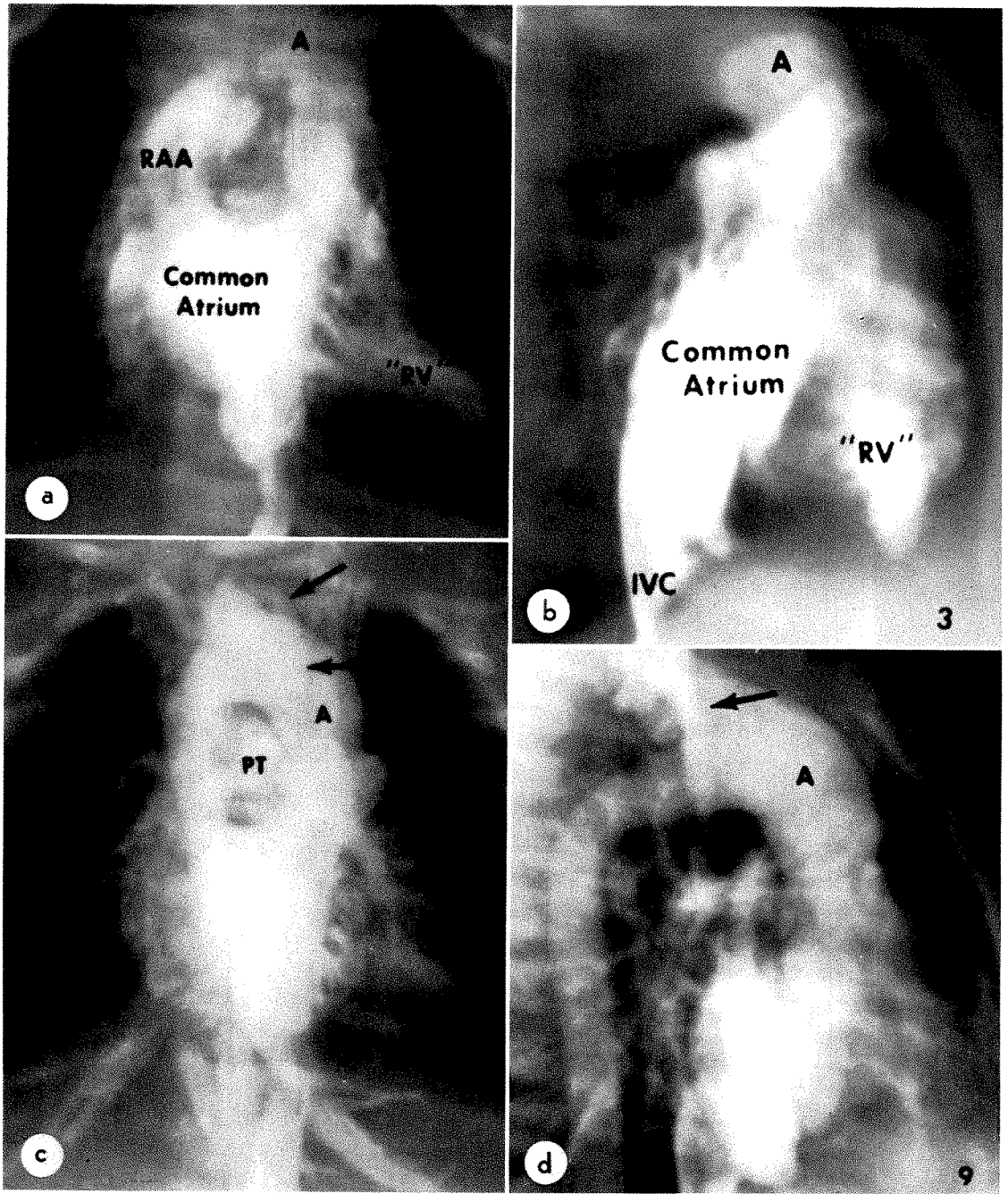


FIG. 3. (*a* and *b*) Frontal and lateral views, respectively. The common atrial chamber is now outlined. The elongated, pointed common ventricle ("RV") is well outlined. The end-on view of the tail of the common ventricle is well seen in the lateral view. The ascending aorta (A) is filled with contrast material. No evidence of opacification of the pulmonary vasculature. (*c*) Frontal view. The enlarged right-sided aorta is now well filled with contrast material. The origin of the aorta arises along the left upper border of the heart and the pulmonary trunk (PT) lies medial to the aorta, both features seen in corrected transposition. Arrows point to a large patent ductus arteriosus which arises from the left subclavian artery and connects with the left pulmonary artery. (*d*) Lateral view, later in the study, shows the aorta to be anterior in location. The arrow points to the patent ductus arteriosus.

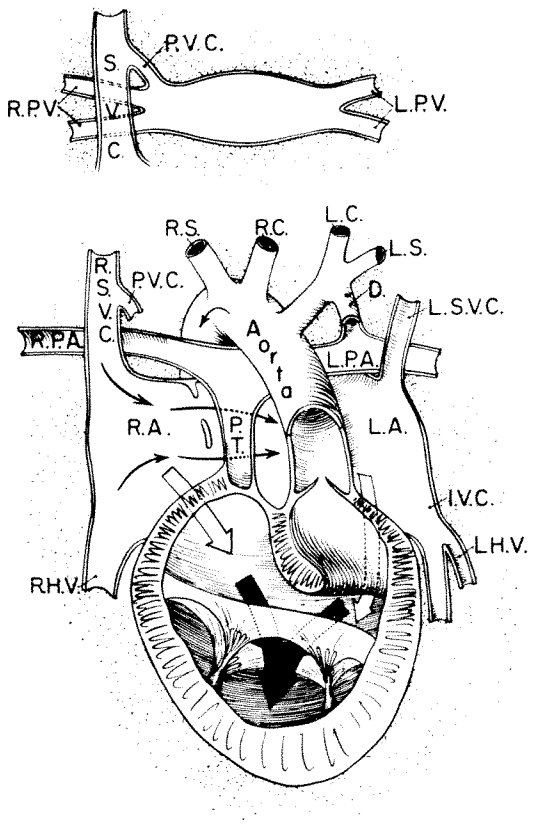


FIG. 4. Cardiovascular anomalies observed at necropsy. The essential findings were a common atrium, a common ventricle, pulmonary valvular atresia, transposition of the great vessels, right aortic arch and systemic and pulmonary venous anomalies. Top line drawing shows the confluence of the pulmonary veins from which the pulmonary venous blood was carried anomalously to the right superior vena cava.

ventricle, there was an infundibular-like pocket from the upper end of which the aorta arose. The pulmonary valve, which was atretic, lay above the posterior aspect of the base of the common ventricle.

Pulmonary venous anomalies were also present. None of the pulmonary veins joined the heart directly. Instead, the pulmonary veins converged behind the heart to form a venous recess. From the right upper aspect of the latter, a vein extended to the right superior vena cava.

COMMENT

The diagnosis of asplenia may be suggested from the frontal roentgenogram of the thorax and abdomen. The horizontal orientation of the lower lobe of the liver[2] associated with malposition of the heart and/or viscera, an enlarged aortic density, a somewhat triangular appearance to the apex of the heart and decreased pulmonary vasculature are characteristic findings.

The common cardiac malformations associated with asplenia usually take the form of either corrected transposition of the great vessels with 2 ventricles, or common ventricle with transposition of the great vessels with inversion of the infundibulum (corrected transposition with rudimentary arterial ventricle).[4] The case reported here is an example of the latter type.

The angiocardiographic appearance of the right-sided venous ventricle in both types of cases is similar. On frontal views this chamber shows itself as an elongated, triangular cavity which occupies the apical region of the heart (Fig. 3*a*).

In the lateral projection, the end-on view of the contrast-filled "tail" of this chamber may be seen as an oval area of increased density lying in an antero-inferior position (Fig. 3*b*).

Forward angiocardiography from a saphenous vein is the diagnostic procedure of choice and will usually define the majority of the vascular and intracardiac anomalies.

Anomalies of pulmonary venous return usually represented by an anomalous connection of pulmonary veins to the right side of the common atrium or to a systemic vein are difficult to demonstrate. Because of the frequent association of pulmonary stenosis or atresia, the major portion of the contrast material is shunted into the systemic circulation, a situation which does not allow adequate definition of the pulmonary venous vasculature. For the same reason it is doubtful that selective ventriculography would provide any better definition than venous angiocardiography. The complex nature of the intracardiac malformations usually precludes surgical correction, and use of selective ventriculography in an attempt to define the pulmo-

nary venous anomaly is probably not warranted.

SUMMARY

The pathologic, roentgenographic and angiocardiographic features in a case of agenesis of the spleen with congenital cardiac disease are presented.

Angiocardiographically, the following anatomic states were established: (1) transposition of the great vessels, (2) pulmonary atresia, (3) common atrium, (4) anomalies of systemic veins, and (5) right aortic arch. Necropsy demonstrated these anomalies as well as a common ventricle, absence of the coronary sinus and total anomalous connection of the pulmonary veins to the right-sided superior vena cava.

The association of multiple complex cardiac anomalies, as observed roentgenologically in this case, suggests the syndrome of agenesis of the spleen and congenital cardiac disease.

Symmetry of the 2 main lobes of the liver giving a horizontal orientation to the lower hepatic edge in the presence of multiple cardiac anomalies, including obstruction to pulmonary arterial flow, adds roentgenologic support for a presumptive diagnosis of asplenia.

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INFRAPULMONARY EFFUSION IN TRAUMATIC RUPTURE OF THE SPLEEN*

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A RUPTURED spleen is a frequent complication of trauma to the lower left chest and left upper abdomen. Despite a high level of suspicion, the condition may not be recognized immediately; indeed, diagnosis may be extremely difficult when there is delayed rupture.[1,2,13] When the lacerated spleen is not diagnosed, the clinical course may end in a sudden marked state of vascular collapse.

REPORT OF A CASE

CASE 1. This was the first hospital admission of an 11 year old boy with the chief complaint of tachypnea, abdominal and chest pain and anorexia.

The child had been well until 3 days prior to admission when he struck his left lower rib cage and left arm on a fence while sledding. He had immediate local pain but no apparent loss of consciousness. He was seen later that night because of abdominal discomfort and roentgenograms of the chest and abdomen taken at another hospital were reported normal. He returned home. Subsequently, he developed dyspnea, cough, some productive sputum, anorexia, chest and abdominal pain. One day prior to admission he developed difficulty in walking and sitting because of rib cage and upper abdominal discomfort. Breathing was limited by a pain in the lower left chest. Physical examination revealed a temperature of 100° F., respirations 40, blood pressure 130/70. The patient appeared in mild respiratory distress with tachypnea but alert and responsive. There was ecchymosis over the anterior portion of the left arm and the left lateral rib cage. Examination of the chest revealed dullness to percussion throughout the left chest, most pronounced at the left base. Examination of the abdomen revealed generalized tenderness with guarding and tenderness in the left upper quadrant. Bowel sounds were absent.

A roentgenogram of the chest revealed pneumonia of the right upper lobe with partial atelectasis of the right upper lobe. There was haziness over the left hemithorax and either elevation of the left hemidiaphragm or a rather large infrapulmonary effusion. There were no rib fractures (Fig. 1).

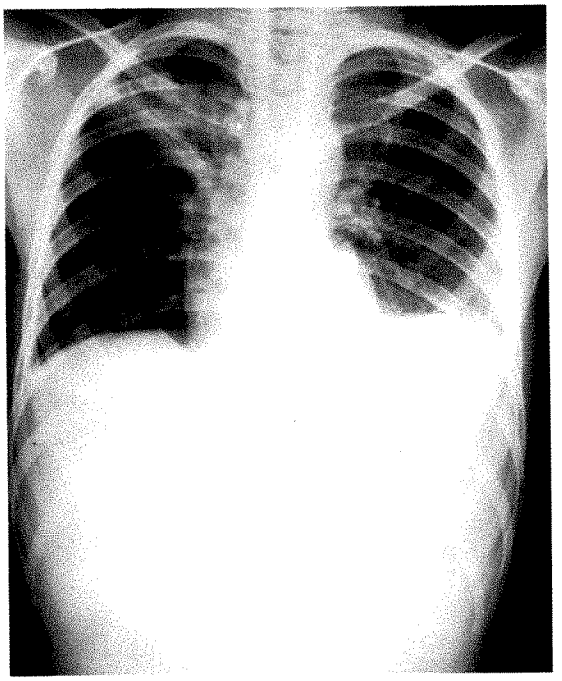


FIG. 1. Case 1. Chest roentgenogram showing pneumonia and partial atelectasis of the right upper lobe. There is haziness over the left hemithorax and either elevation of the left hemidiaphragm or a rather large infrapulmonary effusion. There are no obvious rib fractures.

An abdominal roentgenogram revealed no displacement of the colon or abnormal subdiaphragmatic mass. The splenic outline was not clearly seen (Fig. 2).

Lateral decubitus roentgenograms of the chest showed a large amount of fluid layered out along the lateral chest wall in the left hemithorax, thus establishing the diagnosis of infrapulmonary effusion (Fig. 3).

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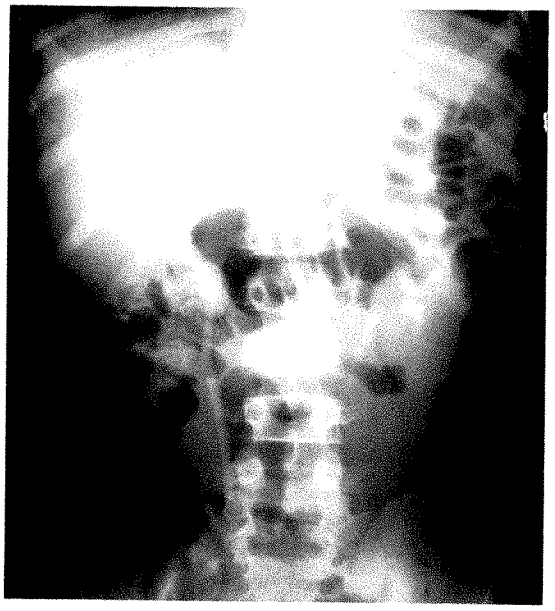


FIG. 2. Case 1. Abdominal roentgenogram showing no displacement of the colon. No obvious subdiaphragmatic mass is noted. The splenic outline is not clearly seen.

The patient was taken to the operating room and the left hemithorax was aspirated. Four hundred cubic centimeters of blood tinge serous fluid was obtained. Bronchoscopy revealed a mucopurulent plug in the right upper lobe bronchus. The abdomen was opened and contained 750 cc. of old blood in addition to multiple clots. There was a large splenic laceration extending to the hilus. The left kidney appeared normal to palpation. The spleen was removed. Postoperatively, the patient progressively improved. The effusion on the left did not reaccumulate. No rib fracture was seen in follow up chest examinations. The right upper lobe atelectasis and pneumonia gradually resolved. On the 18th postoperative day the patient was discharged from the hospital.

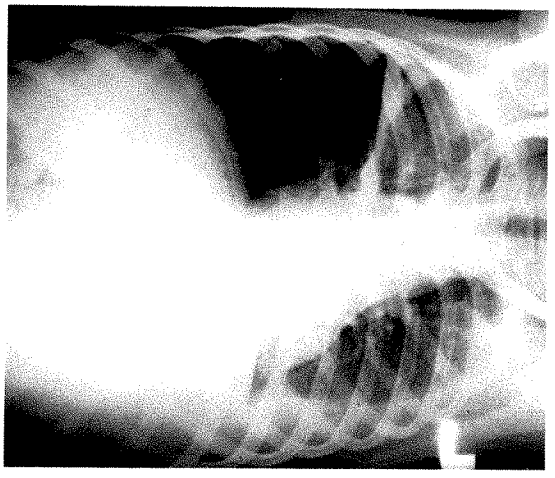


FIG. 3. Case 1. Lateral decubitus roentgenogram of the chest showing a large amount of fluid layered out along the lateral chest wall in the left hemithorax, thus establishing the diagnosis of infrapulmonary effusion.

COMMENT

We have reviewed our last 15 consecutive cases of traumatic ruptured spleen. The results are tabulated in Table 1. One case showed a definite specific diagnostic sign, *i.e.*, enlarged splenic shadow. Eight cases showed definite but nonspecific changes. Six cases showed no significant roentgenographic change.

DISCUSSION

It has been pointed out that roentgenograms of the abdomen and chest can be helpful in the diagnosis of ruptured spleen.[1–13] The following signs have been useful in varying degrees in the past: gastric dilatation, enlargement of the splenic outline, partial loss of splenic shadow, elevation of the left hemidiaphragm, nonvisualization of the spleen,

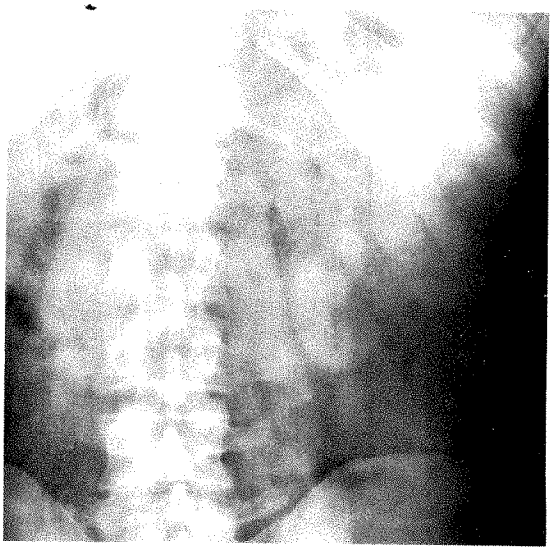


FIG. 4. Case 8. Abdominal roentgenogram showing the enlarged spleen.

TABLE I

Case	Trauma	Roentgen Signs
1. L. A., 11 yr. old male	Ran into fence while playing	Chest roentgenogram: infrapulmonary effusion, right upper lobe pneumonia (Fig. 1 and 3) Abdominal roentgenogram: negative (Fig. 2)
2. C. C., 10 yr. old male	Struck by bike	Abdominal roentgenogram: negative
3. D. H., 17 yr. old male	Wrestling, opponent fell on him	Chest roentgenogram: negative Abdominal roentgenogram: serrated gas pattern in stomach; spleen appears normal
4. P. H., 13 yr. old male	Wrestling	Chest roentgenogram: negative Abdominal roentgenogram: large amounts of gas in large and small bowel
5. L. L., 25 yr. old male	Fell through platform while working	Abdominal roentgenogram: partially obscured psoas borders by intestinal content
6. F. D., 44 yr. old male	Fell on subway stairs	Chest roentgenogram: elevated left hemidiaphragm with hazy pleural reaction Abdominal roentgenogram: slight fullness of splenic shadow
7. J. M., 16 yr. old male	Attacked and beaten	Chest roentgenogram: negative Abdominal roentgenogram: negative Fractured nasal bone
8. D. R., 34 yr. old male	Automobile accident, steering wheel struck chest	Chest roentgenogram: negative Abdominal roentgenogram: spleen enlarged below 12th rib (Fig. 4)
9. P. S., 6 yr. old female	Struck by automobile	Abdominal roentgenogram: ill-defined soft tissue mass left upper abdomen with obliteration of psoas border and left fractured iliac bone
10. M. S., 10 yr. old female	Hit left flank when she fell off sleigh	Chest roentgenogram: negative Abdominal roentgenogram: negative
11. O. W., 36 yr. old male	Automobile accident, steering wheel	Abdominal roentgenogram: slight elevation of left hemidiaphragm. Hazy change over abdomen compatible with fluid
12. R. F., 3 yr. old male	Struck by automobile	Chest roentgenogram: fractured left 7th rib Abdominal roentgenogram: marked amounts of gas in stomach and intestine
13. E. G., 10 yr. old male	Hit tree while sledding	Chest roentgenogram: negative Abdominal roentgenogram: dilated small bowel; gas in colon
14. D. A., 6 yr. old male	Hit abdomen and chest while sledding	Chest roentgenogram: negative Abdominal roentgenogram: negative
15. C. C., 32 yr. old female	Attempted suicide, fell 2 stories	Chest roentgenogram: fractured left 10th and 11th ribs posteriorly Abdominal roentgenogram: dilated stomach

fluid density, gastric displacement, free fluid between loops of bowel, visualization of perisplenic hematoma, serration of the greater curvature of the stomach, diaphragmatic tenting, obliteration of the left psoas outline, and visualization of splenic laceration. All of these have been described in individual reports and series of cases.

Difficulties immediately become apparent. Wyman[12] has pointed out that in a series of 500 routine consecutive abdominal roentgenograms, 55 per cent were technically unsatisfactory to evaluate the splenic shadow and in 30 per cent which were technically adequate, the splenic shadow was not seen. Therefore, the criteria of partial loss of splenic shadow or nonvisualization of the splenic outline seems less dependable. Wyman concluded that the most dependable signs are the downward displacement of the level of the tip of the lower pole of the spleen and the measurable increase in its transverse diameter. This was present in 73 per cent of his series of cases. Wang and Robbins[11] feel that loss of splenic outline and increased density in the area of the spleen are helpful more often than other signs. They conclude that a well outlined small spleen seen on a roentgenogram of good quality practically rules out the diagnosis of ruptured spleen. Recently, Schwartz *et al.*,[7] reviewed 12 cases of traumatic rupture of the spleen in children. They conclude that no roentgen findings or group of findings consistently add up to the diagnosis of splenic trauma. The most significant finding they felt was the exaggeration of mucosal folds on the greater curvature of the stomach, which was present in 7 of their cases. Bollinger and Fowler[1] described left basal pleuritis in patients with delayed splenic rupture. They found this pleuritis with and without rib fractures. The pleuritis appeared 24 hours following trauma.

In Case 1, there was a delay of 3 days between the injury and the visit to the hospital. The chest roentgenogram revealed a shadow in the left lower lung area which could have represented an elevated hemidiaphragm. A decubitus chest roentgenogram, however, revealed a marked infrapulmonary effusion which layered out along the lateral chest wall. A review of the abdominal roentgenogram showed no increase in the spleen size or displacement of gas in the colon. The splenic outline could not be clearly seen. The left kidney was slightly lower than the right. There was no striking ileus nor obvious intra-abdominal fluid. No rib fracture was demonstrated, nor was there any healing fracture seen on subsequent follow up roentgenograms for 3 weeks. A review of our previous cases indicates that the important signs of splenic rupture can often be lacking. In this setting of acute trauma to the left lower chest and left upper abdomen any major roentgenographic abnormality can be important. Rib fracture has been pointed to as an important finding in the chest. Pleuritis has often been observed on delayed roentgenograms. Our case of infrapulmonary effusion adds another nonspecific but potentially important sign of splenic rupture. It is interesting to note here that infrapulmonary effusion can have a strikingly similar appearance to an elevated hemidiaphragm which has been reported frequently in the past.

SUMMARY

We have reviewed 15 cases of splenic rupture due to trauma. An interesting case with infrapulmonary effusion as the major roentgenographic sign is described in detail.

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ROENTGENOGRAPHIC DIAGNOSIS OF SPLENIC ABSCESS

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EARLY preoperative diagnosis of splenic abscess is rarely made. The clinical and roentgenographic signs are vague. A review of the current radiologic literature shows that very little attention has been directed to this entity. The occurrence of what are considered to be classic signs, permitting the preoperative consideration of splenic abscess in a recent case, has prompted a review of the literature and a case report.

ETIOLOGY

Splenic abscess may result from a variety of pathologic processes.

1. Splenic infarction. Here, rapid softening and liquefaction may occur and are seen in sickle cell disease, polycythemia, etc.

2. Trauma. Tissue necrosis may follow splenic injury. Fifteen per cent of the cases reported by Lemmon and Paschal[7] were due to trauma.

3. Mycotic emboli. This may follow as a consequence of bacteremia which results in abscess formation.

4. Local extension. Inflammatory processes involving tissue contiguous with the spleen (pancreas, pleura, bowel) may incite secondary changes in the spleen and cause abscess formation.

5. Primary or idiopathic form. For roentgenologic purposes, liquefactive necrosis of the spleen, with or without secondary bacterial involvement, may be considered as representing a splenic abscess.

INCIDENCE

In Africa, the increased incidence of sickle cell disease parallels the increased incidence of splenic abscess. Gelfand[5] has reported over 40 cases in Northern Rhodesia. Various authors have observed an over-all mortality of between 30 and 100 per cent, but this has diminished somewhat since the onset of the antibiotic era.[1,8,9,10]

REPORT OF A CASE

A 46 year old white male plumber was admitted to Meadowbrook Hospital for the second time on November 23, 1962, with a complaint of abdominal pain and a 25 pound weight loss in the preceding month. His first admission in April, 1961, was for thrombophlebitis of the left leg. Treatment at that time consisted of anticoagulants and symptomatic medication, and he was discharged apparently in good health 3 weeks later.

The patient remained well until February, 1962, at which time he was seen in the Breast Clinic at Memorial Hospital in New York for gynecomastia of the right breast and was followed until October, 1962 with no obvious improvement in this condition.

On October 19, 1962, he was admitted to another hospital for pain in the abdomen. Hepatosplenomegaly was noted. His white blood cell count was 12,000 but he was not febrile. An intravenous pyelogram was reported as demonstrating pressure on the left kidney by a soft tissue mass. He was discharged on October 24, 1962 with little improvement and entered our institution one month later.

Physical examination at this time revealed the liver to be enlarged and the spleen to be palpable and tender. Temperature was 101.4°F. White blood cell count was 27,500. Five blood cultures in 2 days revealed only *Staphylococcus albus*, coagulase-negative (which was felt to be a contaminant) on one occasion.

Roentgenograms on admission showed a diffuse haze in the left upper quadrant of the abdomen with hepatomegaly and splenomegaly, as well as a collection of air below an elevated left hemidiaphragm. Gastrointestinal series demonstrated paradoxic motion of the left hemidiaphragm and displacement of the

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stomach medially by a soft tissue mass. A collection of air in the soft tissue mass was noted to be outside the bowel, and a diagnosis of splenic abscess was suggested.

On November 28, 1962 operation was performed and the spleen was found to be greatly enlarged, filling most of the left upper quadrant. The colon was displaced inferiorly and the stomach medially. Fifteen hundred cubic centimeters of creamy purulent material were drained from the spleen, and it was noted that the capsule was adherent to the diaphragm. A splenectomy was performed. Cultures and smears from the spleen revealed no growth.

The pathology report was (1) acute and chronic necrotizing splenitis probably secondary to infarction and (2) hemosiderosis.

The patient did well postoperatively and was discharged after 25 days to be followed in the Surgery Clinic.

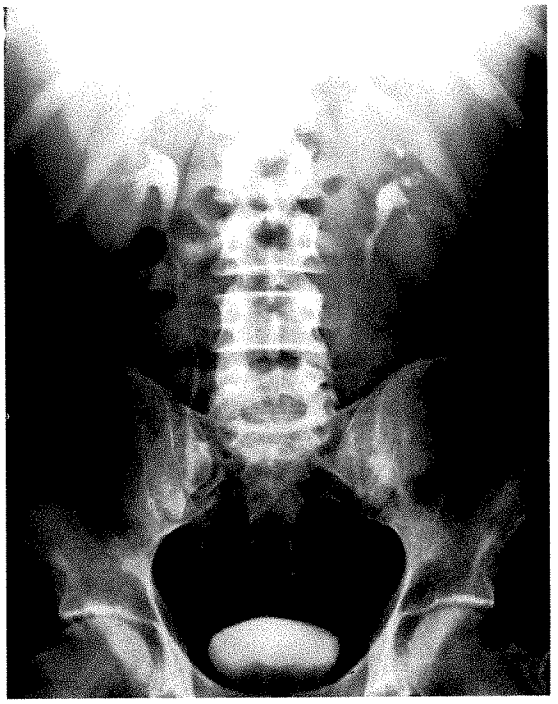


FIG. 2. Intravenous excretory pyelogram shows a soft tissue density in the left upper quadrant pulling the left kidney inferiorly. This occurs via traction on the lienorenal ligament by the enlarged spleen.

ROENTGEN SIGNS

Roentgenographically the following signs of splenic abscess have been reported.

1. Soft tissue mass in the left upper quadrant on the plain roentgenogram of the abdomen. This enlarged mass will be more rectangular and less elongated than the usual tongue shaped spleen noted with splenic enlargement due to other disease (Fig. 1).

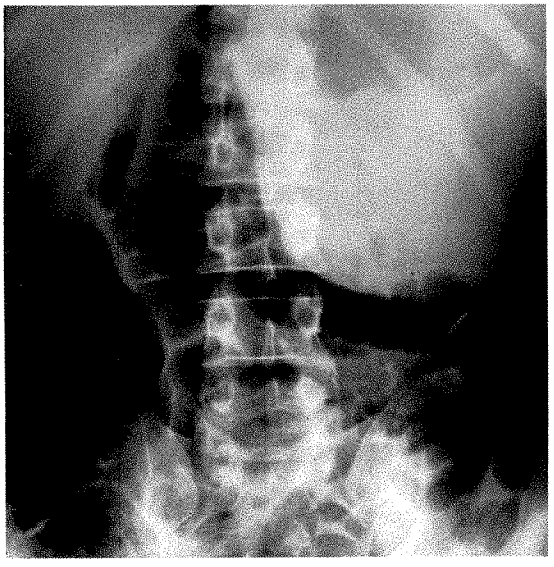


FIG. 1. Soft tissue mass containing an air pocket displacing the splenic flexure of the colon inferiorly. Note the rectangular shape of the splenic outline instead of the normal tongue-like configuration.

2. Presence of an extra alimentary collection of gas in the left upper quadrant. This may take the form of small bubbles or an isolated pocket. If liquefactive necrosis has taken place, an air fluid level may be demonstrated.

3. Displacement of the splenic flexure of the colon. This may be detected on the scout roentgenogram with colonic air acting as a contrast medium or by barium enema study.

4. Inferior displacement of the left kidney. This will be seen to best advantage on the intravenous pyelogram, but also may be observed on the scout roentgenogram. This displacement is believed to be due to the pull on the lienorenal ligament by the enlarging spleen (Fig. 2).[3,4]

5. Stomach bubble displaced medially.



Fig. 3. Large splenic soft tissue mass containing an air pocket and displacing the barium filled stomach medially and colon inferiorly.

Gastrointestinal series and flat roentgenogram of the abdomen will reveal this finding. The gas pocket within the abscess may be easily mistaken for the fundic air shadow; however, the soft tissue thickness between the diaphragm and the gas pocket is usually greater than that seen between the diaphragm and the fundus (Fig. 3).

6. Elevation of the left hemidiaphragm which may or may not displace the mediastinum (Fig. 4).

7. Left pleural effusion. This is a reactive pleural effusion indicating subdiaphragmatic inflammatory disease.

DISCUSSION

It is obvious that no single sign described above is characteristic of splenic abscess. In the presence of several such roentgen findings, correlated with the clinical picture, splenic abscess must be included in the differential diagnosis of left subphrenic inflammatory disease. The condition must be differentiated from subdiaphragmatic abscess, pancreatic abscess, nephritic or perinephritic abscess, and intestinal fistula with local abscess formation.

It is interesting to postulate the reason for the gas shadow seen on roentgenograms, in the absence of any gas producing organisms grown from blood or splenic cultures. Cockshott and Weaver[2] have suggested that oxygen, carbon dioxide and nitrogen are released from the red blood cells and tissue in the process of liquefactive necrosis. The validity of this idea has yet to be conclusively proven. Such a mechanism might explain the air shadow in our own case when all blood cultures were sterile.

It is also of interest that the incidence of splenic abscess is high in those parts of the world where sickle cell disease is common.[5,6] This would favor infarction as an important etiologic factor. The development of small splenic infarcts on the basis of thrombosis in vascular channels with hemolysis of red blood cells is not unusual in sickle cell anemia. In the presence of septicemia, such

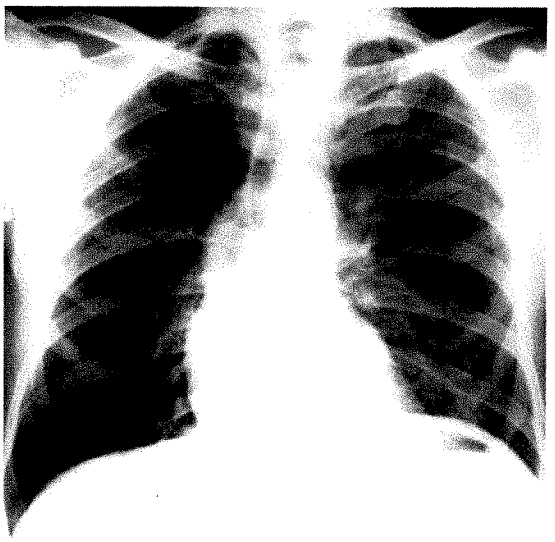


Fig. 4. Posteroanterior roentgenogram of the chest demonstrating slight elevation of the left hemidiaphragm. No pleural inflammatory reaction was seen in this case.

infarcted areas become rather common sites for abscess formation.

SUMMARY

1. The etiology and pathology of splenic abscess are reviewed.

2. An interesting case is reported.

3. The roentgen criteria for the diagnosis of splenic abscess are discussed.

4. In spite of the rarity of this condition, familiarity with the signs and symptoms on the part of the radiologist will allow for a preoperative diagnosis in some cases.

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RADIODENSITIES OF THE SPLEEN ASSOCIATI WITH THALASSEMIA-S DISEASE*

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IN 1929 Bennett[1] first described calcium and iron "incrustations" in the spleen and associated them with sicklemia. Subsequently, areas of increased density in the spleen in conjunction with sicklemia were visualized roentgenographically and reported by Macht and Roman,[6] Ehrenpreis and Schwinger,[2] Jacobson and Zucherman[5] and recently by Hemley *et al.*[4] The purpose of this communication is to report similar roentgenographic findings in a white male with thalassemia-S disease.

REPORT OF A CASE

The patient was a 26 year old white male with a life-long history of recurrent hemolytic crises and persistent anemia despite more than 100 transfusions of blood. During the previous 5 years, the patient's illness had been complicated by episodes of congestive heart failure, and increasing severity of anemia and hepatosplenomegaly. The parents and three siblings are not clinically affected by anemia. One brother had his spleen removed during childhood for a condition similar to that of the patient. Electrophoresis of the patient's hemoglobin demonstrates the A-S pattern with 12 per cent fetal hemoglobin present. The father has A-S hemoglobin and the mother an increased amount of fetal hemoglobin. These findings were believed to substantiate a diagnosis of thalassemia-S disease.

Roentgenographic examination at the age of 18 years demonstrated a moderate enlargement of the patient's spleen, with diffuse mottling and a reticular pattern of increased density. At the time of the present admission, the spleen had increased further in size and roentgenographic density (Fig. 1). Because of lack of response of the patient's hemolysis to medical management, splenectomy was elected. A 1,750 gm. spleen was resected which was covered by a translucent yellowish-white capsule w herent fibrous tags. The sectioned surf brown-red and traversed by innumera white strands, cutting with increased res Microscopically, there were large dep hemosiderin and of calcium salts. The varied markedly in nature. Often there crustation of the supporting tissues, w

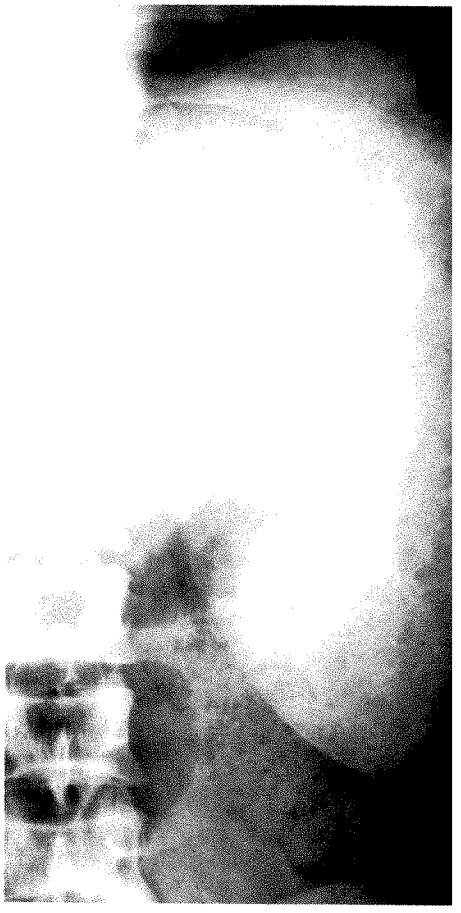


FIG. 1. A roentgenogram of the patient's l men at the age of 26 years demonstratin splenic enlargement and a diffuse and increased splenic density.

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cium salts predominating. In other foci there were large collections of irregular yellow plates of hemosiderin, or collections of brown hemosiderin granules. A small amount of calcium salts was associated with such foci. In the fibrous tissue around the hemosiderin plate and granule collections were multinucleate giant cells of the foreign body type. The areas of fibrosis associated with the mineral collections were broad and irregular in outline and there was focal hemorrhage (Fig. 2). Hemorrhage in the red pulp occurred also. The capsule was thick and hyaline. Roentgenograms of the gross specimen (Fig. 3) and tissue slices (Fig. 4) demonstrated a reticular pattern of increased density with interspersed fine (less than 1 mm.) punctate areas of increased density. In the gross specimen the confluence of the two patterns gave the appearance of coarse (2–5 mm.) nodulation enmeshed in a tangled reticular network.

Using a modification of the method of Peters *et al.*,[7] the spleen from the patient was found to contain 428 μg. of iron per gram of spleen (wet weight). A similar specimen taken from a normal spleen, which was removed following death by accident, showed a content of 107.6 μg. of iron per gram of spleen (wet weight).

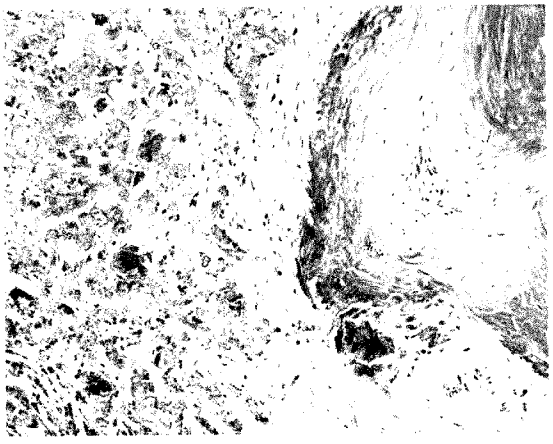


Fig. 2. Photomicrograph of spleen. Calcific material encrusts collagen fibers at the right. Masses of hemosiderin-laden macrophages are in the sinuses of the splenic parenchyma at the left. There is some focal calcification in that portion also (hematoxylin & eosin, 100×).

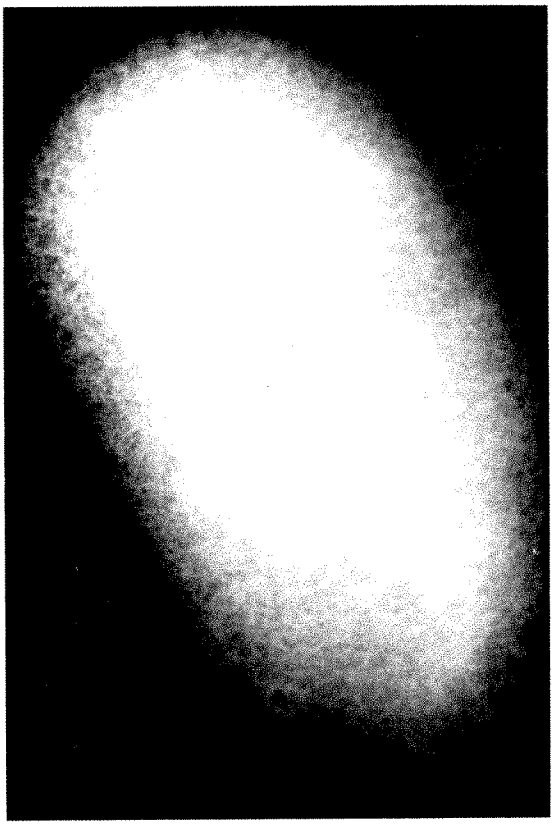


Fig. 3. A roentgenogram of the surgical specimen demonstrating the tangled reticular pattern of increased density.

COMMENT

The differential diagnosis of punctate and diffuse increases in splenic radiodensity has previously been considered.[3] The principal differential of this particular pattern has been considered to lie between sicklemia and thorotrast deposition. Thorotrast can usually be differentiated by history, the normal or small splenic size, and the concurrent opacification of the abdominal lymph nodes and liver.

The pathologic findings differ most strikingly from those of sicklemia not in microscopic detail, but in the large spleen size. Pure sicklemia of this duration is usually associated with splenic atrophy. The foreign body granulomatous response is also unusual in degree. The large spleen size is more in keeping with thalassemia major, sickel cell-hemoglobin C disease, or thalassemia-S disease. In the case of thalassemia major, the microscopic appearances usually differ from those in the present patient.

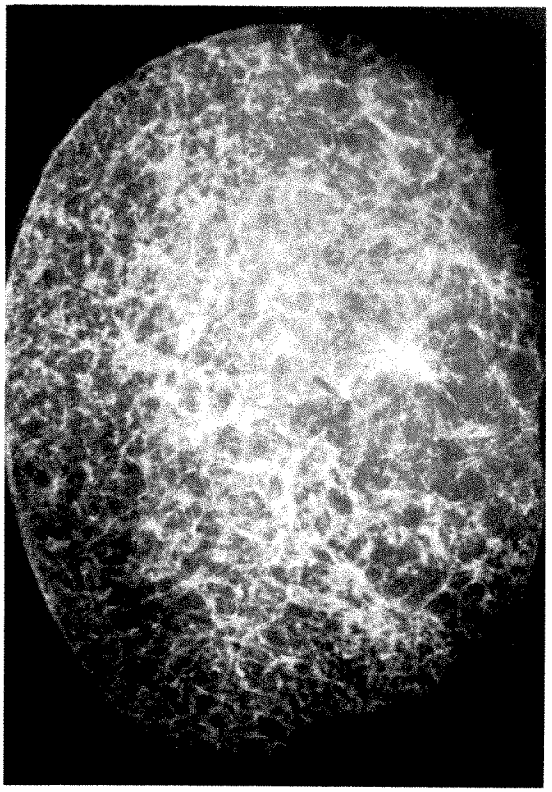


FIG. 4. A roentgenogram of a 2 mm. cross sectional slice of the spleen demonstrating the density to be concentrated in the interstices and septa of the spleen with the lobules spared.

Although the absolute values of splenic iron may not be completely accurate, there is little question that the fourfold difference in iron content noted between the diseased and a normal spleen is significant.

Of the previously reported cases with the clinical diagnosis of sicklemia and splenic opacification, the majority, if not all, have been in the Negro race. In the 3 cases in which the hemoglobin electrophoretic pattern was reported, the homozygous-S characteristic was observed.[4] This case shows that a similar pattern of splenic opacification can occur with thalassemia-S disease and in a person ostensibly of Caucasian extraction.

SUMMARY

Punctate and diffuse increase in the density of the spleen occurring in a case of thalassemia-S disease in a white male was demonstrated on *in vivo* and *in vitro* roentgenograms.

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A ROUTINE SURVEY ROENTGENOGRAM OF THE ABDOMEN ON 500 CONSECUTIVE PATIENTS OVER 40 YEARS OF AGE*

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ALTHOUGH a routine roentgenogram of the chest is generally accepted as an integral component of a complete health evaluation, equal in importance to a complete blood cell count and urinalysis and a close rival of a thorough history and physical examination, there has been little study of the possible benefits to be derived from a routine survey roentgenogram of the abdomen. Even though virtually all radiologists consider a survey roentgenogram as a necessary component of intravenous urography, and many appreciate its value as a preliminary step before contrast studies of the gastrointestinal and biliary tracts, there are no known institutions that obtain such a roentgenogram as a routine admission procedure. Levine and Crosbie[3] believe that this reluctance can largely be attributed to the necessity for the patient to undress more completely than for a chest roentgenogram, to possible confusion resulting from fecal material in the uncleansed intestinal tract, to the radiation exposure involved and to a lack of knowledge concerning the information that can be obtained from such a study. These investigators derived from a routine abdominal roentgenogram valuable information that otherwise might have been overlooked in 11 per cent of a series of 242 consecutive patients admitted to a Veterans Administration Hospital. This figure compares favorably with the approximately 18 per cent of routine admission chest roentgenograms that revealed significant positive findings. These chest roentgenograms were reviewed by the same investigators in the same institution, apparently on similar patients. Since to our knowledge there has been only one additional study[4] of the value of a routine admission roentgenogram of the abdomen, and this in an institution devoted to the care of patients with chest diseases, it seemed desirable to assess the information that could be obtained by such a procedure on a series of patients seen at a University Medical Center treating a general hospital population.

MATERIAL AND METHODS

The first 500 patients over 40 years of age admitted to the inpatient and outpatient services of the newly activated University of Kentucky Medical Center were selected for this study. Patients under 40 years of age were rejected to minimize objections concerning radiation exposure. Two hundred and sixty-eight patients were male and 232 female. Four hundred and seventy were white and the remaining 30 were Negroes.

This Medical Center serves a patient population that is largely indigent. Since a facility such as this previously has not been available to the indigent population of Kentucky, most of the patients seen have serious medical problems. There was a very small percentage of admissions for routine health evaluation. It was expected that a relatively high percentage of positive roentgenograms would be found.

A supine roentgenogram of the abdomen was made when the patient visited the Department of Radiology for routine chest roentgenography. There was no prior preparation of the patient, and no patient was recalled for a repeat study because of

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the quality of the resulting roentgenogram. Although a few of the roentgenograms were not of optimal diagnostic quality, all were thought to be at least acceptable by the viewing radiologists. In addition to noting pathology, kidney and spleen outlines, visibility of the properitoneal lines, psoas shadows and gas in the small bowel were recorded. Note was made of whether a properitoneal line had been cut off the roentgenogram by virtue of positioning or size of the patient.

All of the roentgenograms were studied independently by 2 staff radiologists (H.D.R. and A.L.), and the results were tabulated with automatic data processing equipment. Roentgenograms on which there was disagreement were studied independently by a third radiologist (D.J.H.) with a consensus being finally reached in conference on all roentgenograms with disputed findings.

Dual reading, with arbitration of disputed findings as suggested by Yerushalmy, Harkness, Cope and Kennedy,[5] who studied the results of mass roentgenography of the chest, was adopted to reduce false negatives. It is of interest that initial disagreement was of the same order as indicated by Garland[2] in discussing the accuracy of various diagnostic procedures. There was disagreement as to the presence of significant pathology on the first readings in 22.0 per cent of the roentgenograms. Much of this disagreement stemmed not from observer errors so much as from variations between the 2 radiologists concerning the type of pathology to be considered significant.

The criteria used to designate roentgenographic evidence of pathology as "significant" were quite severe and much more rigid than those employed by Levine and Crosbie[3] or Nishio, Kass and Levine.[4] Phleboliths, degenerative changes of the lumbar spine, arterial calcification (unless suggestive of aneurysm), prostatic calculi, minor congenital anomalies, scoliosis and similar changes were recorded but not tabulated as significant pathology. Such conditions were not felt to add materially to the physician's evaluation of the patient's health.

Finally, the patients' clinical records were reviewed to evaluate agreement between the roentgenographic findings and other clinical data and to determine, if possible, what information is gained from a survey roentgenogram of the abdomen that would be missed with a good baseline history, physical examination and basic laboratory studies. To this end, the total record of each individual (87 patients with 96 diagnoses) with roentgenograhic evidence of significant pathology unsuspected on the clinical evaluation, or suggestive evidence of pathology requiring additional studies, was reviewed in detail by one of us (E.D.P.), a clinician, to determine the adequacy of the clinical data. The records of patients adjudged to have adequate histories, physical examinations and laboratory studies were assessed to determine whether the data were sufficiently suggestive of the condition seen on the survey abdominal roentgenogram to permit a direct diagnosis, or point to the need of additional studies directed toward revealing the condition.

RESULTS

Table 1 shows the over-all results of this study.

Category I (no significant pathology). Approximately two-thirds of the patients showed no significant pathology on the routine abdominal roentgenogram.

Category II (significant pathology consistent with initial clinical evaluation). There were 60 (12.0 per cent) patients with 68 (13.6 per cent) significant roentgenographic findings (Table II) which were consistent with the initial clinical evaluation. The diagnosis would have been forthcoming since an abdominal roentgenogram or additional roentgen examinations were indicated and ordered in the course of the patient's clinical evaluation. The survey film of the abdomen was only of corroborative value in these instances.

Category III (significant pathology

TABLE I

PATHOLOGY DISCOVERED ON A SURVEY ROENTGENOGRAM OF THE ABDOMEN ON 500 CONSECUTIVE PATIENTS OVER 40 YEARS OF AGE

Category	No. of Patients	Diagnoses
I. No significant pathology	327 (65.4%)	327 (65.4%)
II. Significant pathology consistent with initial clinical evaluation	60 (12.0%)	68 (13.6%)
III. Significant pathology missed on survey	9 (1.8%)	9 (1.8%)
IV. Suggestive evidence of pathology ruled out by additional studies	24 (4.8%)	24 (4.8%)
V. Suggestive evidence of pathology requiring additional studies	52 (10.4%)	56 (11.2%)
VI. Significant pathology unsuspected on initial clinical evaluation	35 (7.0%)	40 (8.0%)
Totals	507 (101.4%)*	524 (104.8%)*

* The roentgenographic findings placed some patients in more than one category, and in some patients more than one diagnosis in a single category was made. This accounts for the total percentages exceeding 100%.

missed on survey). There were 9 (1.8 per cent) instances in which significant pathology was missed. These are true "false negatives" since review of the abdominal roentgenogram, after perusal of the patients' records, showed definite pathology that was not observed on the dual reading (Table III). One error (a pelvic mass) was not clearly visible on the survey roentgenogram because of less than optimal roentgenographic technique. The roentgenogram of the patient with Paget's disease was interpreted as showing a few small osteoblastic metastases. It is evident that difficulty was encountered in identifying some pelvic masses.

Category IV (suggestive evidence of pathology ruled out by additional studies). Seventy-six roentgenograms (Categories IV and V) revealed enough evidence suggestive of significant pathology to warrant additional clinical studies. In 24 (4.8 per cent) instances (Category IV) sufficient studies were performed in the course of the patients' evaluation to rule out the pathology suggested. These findings are listed in Table IV and in a sense represent justifiable "false positives." These "fales positives" are not true errors since the roentgeno-

TABLE II

SIGNIFICANT PATHOLOGY CONSISTENT WITH INITIAL CLINICAL EVALUATION

Category II

Roentgenographic Diagnosis	No. of Positive Roentgenograms
Adynamic ileus	15
Renal lithiasis	8
Hepatomegaly	6
Splenomegaly, hepatosplenomegaly, ascites	4 each
Cholelithiasis, heavy metal therapy	3 each
Enlarged kidney, abdominal aortic aneurysm, spinal fusion, old pelvic injury, malum coxae senilis	2 each
Retroperitoneal mass, toxic colitis, uterine fibroid, pathologic fracture of the lumbar spine (from metastatic malignancy), calcification in a malignant tumor, bullet, protrusio acetabuli, osteomyelitis, osteitis pubis, neuropathic joint disease, severe localized intervertebral disk disease (possibly infectious)	1 each

TABLE III

SIGNIFICANT PATHOLOGY MISSED ON SURVEY

Category III

Missed Roentgenographic Diagnosis	No. of Positive Rentgenograms
Pelvic mass (one due to poor roentgenographic technique)	4
Perinephric abscess, hepatomegaly, ankylosing spondylitis, ascites, Paget's disease	1 each

TABLE IV

SUGGESTIVE EVIDENCE OF PATHOLOGY RULED OUT BY ADDITIONAL STUDIES

Category IV

Roentgenographic Diagnosis	No. of Positive Rentgenograms
Renal lithiasis	5
Ureteral lithiasis	4
Abdominal aortic aneurysm	3
Adynamic ileus, splenomegaly	2 each
Pelvic mass, left upper quadrant mass, right upper quadrant abscess, small kidney, cholelithiasis, adrenal calcification, abnormal intestinal gas collection, compression fracture of a vertebral body	1 each

graphic evidence, although not conclusive, was sufficiently convincing to require the additional investigation.

Category V (suggestive evidence of pathology requiring additional studies). An additional 52 (10.4 per cent) patients in 56 (11.2 per cent) instances (Table v) revealed sufficient evidence of significant pathology to warrant additional studies which as yet have not been performed. It should be noted that the clinical services did not have the benefit of the findings on the abdominal roentgenogram unless such was ordered as part of the patient's general study. There can be no doubt that many of these patients harbor the suggested disease process, but it is impossible to know at this time the exact percentage since many are either dead, unavailable, too old or too infirm to justify further investigation.

TABLE V

SUGGESTIVE EVIDENCE OF PATHOLOGY REQUIRING ADDITIONAL STUDIES

Category V

Roentgenographic Diagnosis	No. of Positive Roentgenograms
Renal lithiasis	9
Ureteral lithiasis	7
Cholelithiasis	6
Adrenal calcification, abdominal aortic aneurysm	4 each
Small kidney, unusual abdominal calcification	3 each
Enlarged kidney, splenomegaly, unusual liver calcification, unusual pelvic calcification	2 each
Foreign body in liver, foreign body in pelvis, hepatomegaly, enlarged gallbladder, pelvic mass, retroperitoneal mass, splenic artery aneurysm, benign lytic lesion of the pubis, sacroiliac arthritis, severe localized L4–L5 degenerative disk disease, infarct of the femoral head, severe spina bifida (with dribbling of urine)	1 each

Category VI (significant pathology unsuspected on initial clinical evaluation). There were 35 (7.0 per cent) patients with 40 (8.0 per cent) findings, shown in Table VI, of significant pathology which was not suspected at the initial examination of the patient. Although some of these diagnoses were established by the clinical services in due time, most of the patients were discharged without such diagnoses being entertained. Nevertheless, it is felt that the roentgenographic evidence is sufficiently convincing to warrant the diagnoses even in

TABLE VI

SIGNIFICANT PATHOLOGY UNSUSPECTED ON INITIAL CLINICAL EVALUATION

Category VI

Roentgenographic Diagnosis	No. of Positive Roentgenograms
Cholelithiasis	11
Renal lithiasis	8
Previous heavy metal therapy	5
Splenomegaly, hepatomegaly, metastatic carcinoma of prostate	2 each
Hepatosplenomegaly, hepatic artery aneurysm, splenic artery aneurysm, surgical needle in abdomen, eventration of left leaf of diaphragm, broken nail in hip, small kidney, gastric atony, ankylosing spondylitis, uterine fibroid	1 each

Table VII

FREQUENCY OF IDENTIFICATION OF PROPERITONEAL LINES, KIDNEY OUTLINES, AND PSOAS SHADOWS

	Properitoneal Lines	Kidney Outlines	Psoas Shadows
Both definitely seen	277 (55.4%)	108 (21.6%)	277 (55.4%)
Only one definitely seen	135 (27.0%)	173 (34.6%)	131 (26.2%)
Neither definitely seen	88 (17.6%)	219 (43.8%)	92 (18.4%)
No. of initial disagreements	139 (27.8%)	143 (28.6%)	105 (21.0%)

those cases where clinical corroboration was not obtained.

Evaluation of the presence of properitoneal lines, kidney outlines and psoas shadows are tabulated in Table VII. In 122 (22.4 per cent) roentgenograms, at least one properitoneal line was cut off due to poor positioning or the size of the patient. The frequency with which small bowel gas and the splenic outline was observed is shown in Table VIII.

Results of the review of the patients' clinical records are shown in Table IX. A significant number of records revealed the history, physical examination and laboratory data to be inadequate to enable the clinician to suspect the suggested abnormality. These patients were seen either in the emergency room as outpatients or were admitted for surgery. In these instances, the clinician's attention was obviously focussed on the problem at hand. It is of special interest that in those patients with adequate evaluation, the history, physical examination and laboratory data would not have led to the condition discovered on the survey abdominal roentgenogram in approximately 75 per cent of the individuals studied.

COMMENT

The results of this study suggest that a routine survey roentgenogram of the abdomen approaches or equals the value of a routine chest roentgenogram as a source of unsuspected pathology. Since the many reported mass surveys with chest roentgenograms have been on population samples vastly different from our patients, the written reports of the initial chest examination (posteroanterior and lateral) of these 500 individuals were reviewed. Forty-one per cent of the chest roentgenograms demonstrated evidence of significant pathology. This figure is comparable to the 32.8 per cent of abdominal roentgenograms showing positive evidence of similar significance.

Only 1 case of degenerative disease of the lumbar spine was accepted as significant. In this instance the appearance raised the possibility of an infectious process. It was felt at the outset that determination of symptomatically "significant" degenerative disease of the lumbar spine was not dependable from a roentgenogram of the abdomen. Undoubtedly, some instances of severe degenerative spinal disease were significant, and had they been included, the percentage would have equaled or exceeded 41 per cent. Well over 90 per cent of the patients showed some degenerative spinal changes. There were 27 roentgenograms that showed distinct narrowing of an intervertebral space despite the fact that L5-S1, and often L4-L5, are poorly projected for this purpose. Ten other roentgenograms, not included in the tables, were recorded as showing osteoporosis. Levine and Crosbie[3] and Nishio, Kass and Levine[4] accepted such

Table VIII

FREQUENCY OF IDENTIFICATION OF SPLENIC OUTLINE AND SMALL BOWEL GAS

	Spleen	Small Bowel Gas
Definitely seen	86 (17.2%)	146 (29.2%)
Not definitely seen	414 (82.8%)	354 (70.8%)
No. of initial disagreements	72 (14.4%)	155 (31.0%)

TABLE IX

REVIEW OF CLINICAL EVALUATION OF PATIENTS IN *Categories V and VI*
(87 patients with 96 diagnoses)

	Inadequate	Adequate	Adequate Clinical Evaluation	
			Suggestive of Diagnosis	Not Suggestive of Diagnosis
History	25 (26.1%)	71 (73.5%)	20 (28.2%)	51 (71.8%)
Physical Examination	28 (29.2%)	68 (70.8%)	13 (19.1%)	55 (80.9%)
Laboratory Data	31 (32.3%)	65 (67.7%)	15 (23.1%)	50 (76.9%)

findings as indicating significant disease. It is of some interest that abnormal arterial calcification was recorded on 204 occasions, and phleboliths were noted on 409 studies.

It is clear that a routine survey roentgenogram of the abdomen will demonstrate an appreciable number of disease processes that are missed with the usual thorough histories, physical examinations and laboratory studies performed by both, house and attending physicians in a University Hospital. This study revealed that renal stones, gallstones, and abdominal aneurysms are frequently, if not usually, silent. Findings of this nature have significant implications for the patient's future medical care. Careful follow-up of selected individuals from this group may well contribute to our knowledge of the natural history of entities such as renal stones and abdominal aneurysms.

The data of this survey indicate that absence of one or both renal or psoas outlines, as well as the properitoneal fat lines, is frequent and should be considered of no significance without confirmatory findings of disease (Table VII). Elkin and Cohen[1] came to a similar conclusion concerning the psoas shadows after reviewing roentgenograms of 200 patients with particular emphasis being placed on the appearance or absence of the psoas outlines. Kidney outlines were distinctly less frequently seen than psoas shadows or properitoneal lines. The frequency with which small bowel gas could be identified (Table VIII) was surprising. Although this figure (29.2 per cent) includes cases in which small bowel gas was indicative of disease, it is obvious that gas often can be seen in this location without being pathologic.

It is evident from Table X, as expected, that the percentage of abdominal roentgenograms showing significant disease increases with the age of the patient. This consideration, in addition to the reduced hazard of radiation induced genetic damage, suggests that survey roentgenograms

TABLE X

EVIDENCE OF PATHOLOGY RELATED TO AGE

Age	Total Patients	*Category V* Suggestive Evidence of Pathology Requiring Additional Studies	*Category VI* Significant Pathology Unsuspected on Initial Clinical Evaluation
Less than 50	211 (42.2%)	14 (6.6%)*	5 (2.4%)*
50–60	159 (31.8%)	15 (9.4%)*	11 (7.0%)*
60–70	90 (18.0%)	17 (18.8%)*	9 (10.0%)*
Over 70	40 (8.0%)	6 (15.0%)*	10 (25.0%)*
Totals	500 (100.0%)	52 (10.4%)	35 (7.0%)

* These percentages relate to the total number of patients in this age group.

of the abdomen should be restricted to individuals beyond the age of 40 years.

This survey not only has been of value in revealing unsuspected pathology and identifying patients to be recalled for needed additional studies but also has distinctly sharpened the diagnostic acuity of the participating radiologists. Individual blind spots (splenic outlines, renal stones, etc.) were identified with edifying results and further emphasize the need for dual or triple readings (preferably by different individuals) in studies of this nature. It became clear that, for a positive diagnosis, calcification must be seen in both walls of an abdominal aortic aneurysm since a single curvilinear line of calcification well to the left of the location of a normal aorta may represent only aortic tortuosity.

Although the benefits of a survey such as this are significant, it represents a prodigious amount of labor which would be nearly unapproachable without data processing equipment. It is worth emphasizing that the information being sought should be clearly defined before an investigation of this nature is started, and the usual inclination to broaden the project should be sternly rejected.

SUMMARY

A routine survey roentgenogram of the abdomen on the first 500 patients over 40 years of age admitted to the outpatient and inpatient services of the University of Kentucky Medical Center were studied independently by 2 radiologists. Discovered pathology and frequency of visualization of various normal structures were recorded. Disagreements were arbitrated in conference with a third radiologist. Clinical records were reviewed by the radiologists and a clinician to determine whether the suggested pathology would have been, or should have been, discovered or suggested in the initial clinical evaluation of the patients.

The results indicate that a roentgenogram of the abdomen is comparable in value to a chest roentgenogram as a survey procedure to uncover pathology in a group of patients such as those included in this study. Seven per cent (35) of the patients revealed 40 roentgenographic diagnoses of significant pathology unsuspected on the initial clinical evaluation. In 10.4 per cent (52) additional patients, roentgenographic evidence of pathology was sufficiently suggestive of 56 diagnoses to require additional studies. These 96 conditions discovered on the abdominal roentgenograms were not suspected in approximately 75 per cent of those patients adjudged to have had adequate histories, physical examinations and laboratory studies. Equivocally significant entities such as degenerative disease of the lumbar spine are not included in these totals. There were 9 (1.8 per cent) instances of significant pathology missed on the survey. Absence of one or both kidney outlines, psoas shadows or properitoneal lines is quite common and of no significance without additional confirmatory positive findings.

It is apparent from this study that in a population admitted to a general hospital, a routine survey roentgenogram of the abdomen on all individuals over 40 years of age will reveal a significant number of abnormalities which would not be detected even with a good general evaluation.

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THE DEVELOPMENT AND USE OF A MAMMOGRAPHIC TECHNIQUE*

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ROENTGENOGRAPHIC examination of the breast has been employed for many years in many countries by many physicians but usually in a more or less sporadic fashion. A few investigators, however, sustained a special interest in the procedure, making contributions of various kinds and value toward its development.[2,4,5,6,8,9,11,13,15] These efforts plus the crucial fact that surgeons now are receptive to any procedure that might favorably change the cure rate of cancer of the breast, a figure little altered for years, have generated a widened investigation regarding the value of mammography.

Confusion has arisen among radiologists due to the differences in recommended roentgenographic techniques, the difficulties encountered in executing certain ones, the complexities of roentgenologic identification of diseases of the breast, and the varying degrees of clinical effectiveness as reported by different statistical methods. For these reasons an investigative program was begun at this institution embodying all aspects of mammography.

The first phase of this project has been completed and its results have been applied in over 2,000 examinations. The present report gives the methods employed and the results produced which have made possible the development of a mammographic technique suitable for use by all radiologists.

Low kilovoltage and fine grain film are inescapable requirements for satisfactory mammographic work. Since in this kv. range space charge effects often reduce the tube current below the value on the ma. setting, the kv. meter frequently underestimates the actual kilovoltage employed. Some of the techniques that have been published fail to take this into account. Damage to the roentgen-ray tube can be a real hazard, for the long exposures necessary to obtain good mammograms make burn-up of the target a possibility. In addition, the filament current may be raised to a value in excess of the tube capacity, leading to a danger of filament burn-out. Also, at low kv. absorption of the roentgen rays by tissue is relatively high, producing great variations in the density of the exposed film with slight changes in the composition and size of the breast. Because of such facts as these, the skin dose is higher than in other diagnostic examinations and with some techniques it can constitute a hazard to the patient.

MEASUREMENTS

TECHNICAL FACTORS

True ma. was measured by the panel ma. meter; kvp. was measured by a specially calibrated primary voltmeter and checked by spark gap, precision voltage divider and oscilloscope, high-voltage rectifier and electrostatic voltmeter, and spectra were obtained by computer analysis of absorption curves.[7,10] Time was measured by an electric stop clock. Thus the reported values of mas. and kvp. are true.

DIAGNOSTIC QUALITY

In this evaluation of mammographic techniques, it was our aim to define objectively for mammography all significant factors which enter into diagnostic quality and to devise quantitative and reproducible measurements of these factors.

Contrast. As used here, contrast is a

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measure of the ability to separate by their differences in film density the various tissues which comprise the breast. At the low kv. employed in mammography, the difference in film density is due primarily to the difference in effective atomic number of the absorber. To measure contrast, a special constant-thickness step wedge was designed in which the absorber varied from 100 per cent water ($\overline{Z}=7.5$) to 100 per cent polystyrene ($\overline{Z}=5.8$), the polystyrene being very nearly fat-equivalent. The slope of the linearized curve of optical density *vs.* percentage polystyrene was taken as a measure of the contrast, and was called the "atomic number contrast."

Density. The over-all film density produced was defined as the density underneath the fat-equivalent (polystyrene) and of the linearized Z-step wedge curve described above. This was called the "reference optical density."

Resolution. To define the ability of a technique to resolve minute flecks of calcification, a phantom consisting of a polystyrene block with a number of tapped holes containing from 24 to 80 threads per inch was made. These holes were filled with a saturated solution of calcium nitrate. Films were exposed, and the largest number of threads per inch which could be resolved was noted. This value, "threads per inch resolvable," indicates the degree of resolution of which a technique is capable.

Latitude. The sensitivity of film density to variation in absorber thickness was measured by metering the density variation of a film exposed under a composite lucite-polystyrene step wedge. This variation, the "thickness dependence," has been expressed as the per cent change in mas. necessary, per cm. change in absorber thickness, to maintain a constant optical density. It is an inverse measure of the ability of a technique to demonstrate on a single film both the internal breast structures, where the absorber is thick, and the skin where the absorber is thin.

Tube cooling time. The tendency toward tube overheating was measured by determining the minimum safe cooling time between repeated identical exposures. This, of course, varies with the tube employed; reported values are for a Dynamax 50.

Skin dose. This was measured by a Memorial Hospital diagnostic x-ray chamber,[3] embedded half-way in a paraffin block, using a Baldwin-Farmer electrometer.

Those technical factors whose effects were examined and the range of which was investigated are shown in Table 1. Since to investigate all combinations of the various technical factors would require an impracticably large number of films, a standard technique was set up and the various technical factors were varied, one at a time, from the standard values.

Table I

RANGE OF TECHNICAL FACTORS

Kilovoltage (peak)	25, 30, 35
Added filter (mm. Al)	0, ½, 1
Film type	Non-screen, AA, M*
Target skin distance (inches)	16, 36
Milliampere seconds (for reference O.D.)	1.0, 2.0
Focal spot (mm.)	1, 2
Development time (min.)	5, 10

Standard technique: 30 kvp.; ½ mm. Al added filter; AA film; 16 inch target skin distance; reference optical density = 2; 2 mm. focal spot; 5 min. development.

* See text for other film types checked for suitability.

RESULTS

The various measurements which were made are summarized in Tables II through VI. It is necessary to keep in mind in reading these tables that, unless otherwise specified, the technical factors used are those for the standard technique.

Kilovoltage (peak). As kvp. is increased, the mas. required to expose a film properly decreases sharply and the skin dose also decreases. The latitude improves, but the contrast diminishes. At the same time, the resolution is somewhat degraded, presumably due to an increase in Compton scattering and a decrease in the difference between the absorption coefficients of calcium and soft tissues. A marked improvement in cooling time is noted.

TABLE II

EFFECT OF KILOVOLTAGE (PEAK)

	Kvp.		
	25	30	35
Added filter (mm. Al)	0	1/2	1
Milliampere seconds	2,300	960	350
Skin dose (r)	19	6	2
Atomic number contrast	2.0	1.5	1.1
Thickness dependence (% mas./cm.)	28	22	18
Resolution (threads/inch)	72	64	56
Cooling time (min.)	2.0	0.9	0.3

TABLE III

EFFECT OF ADDED FILTRATION

Kilovoltage (peak)	30	30	30
Added filter (mm. Al)	0	1/2	1
Milliampere seconds	720	960	1,300
Skin dose (r)	10	6	5
Atomic number contrast	1.7	1.5	1.4
Thickness dependence	26	22	20
Resolution	64	64	64
Cooling time (min.)	0.7	0.9	1.2

Filtration. As filtration is increased, the exposure required also increases with an improvement in skin dose. The resolution is not markedly affected, the contrast is slightly reduced, and the cooling time increases. An increase in added filter from zero to 0.5 mm. aluminum results in a substantial reduction in skin dose, but an increase from 0.5 to 1.0 mm. aluminum further reduces the skin dose only slightly. In consequence of these findings, an added filter of 0.5 mm. aluminum was adopted as the standard in addition to the inherent filtration of the tube.

Target skin distance. An increase in the target skin distance from 16 to 36 inches requires about a fourfold increase in mas. and consequently a sixfold increase in cooling time. The skin dose is only slightly reduced but the resolution is improved considerably. It is now evident that the standard target skin distance of 16 inches employed throughout this series of determinations was too small, causing an undue

TABLE IV

EFFECT OF TYPE OF FILM

Film	Non-screen	AA	M	Polaroid
Milliampere seconds	240	960	3,200	10
Skin dose (r)	1.5	6	20	0.06
Atomic number contrast	1.5	1.5	1.5	very small
Thickness dependence	22	22	22	very small
Resolution	48	64	64	24
Cooling time (min.)	0.2	0.9	4.2	0

Table V

EFFECT OF FOCAL SPOT SIZE		
Size of focal spot (mm.)	1	2
Resolution (threads/inch)	72	64
EFFECT OF TARGET SKIN DISTANCE		
Target skin distance (inches)	16	36
Skin dose (r)	6	5
Milliampere seconds	960	3,600
Resolution (threads/inch)	64	80
Cooling time (min.)	0.9	5.7
EFFECT OF INCREASED DEVELOPMENT TIME		
Development time (min.)	5	10
Film type	M	M
Milliampere seconds	1,820	1,050
Reference optical density	2.0	2.0

loss of resolution and in consequence, a greater target skin distance has been employed when patients are examined.

Focal spot size. Reduction of focal spot size from 2 mm. to 1 mm. causes an improvement in resolution. However, the danger of damage to the target is made so great that with industrial films an exposure must be interrupted, the tube allowed to cool, and the exposure resumed. This is of course impractical and it is thought that a comparable improvement in resolution is better gained by increasing the target skin distance as noted above.

Film type. The films most thoroughly evaluated were Non-screen; type AA industrial, which requires about 4 times the exposure of Non-screen film; and type M industrial which requires approximately 3 times the exposure of type AA. There was no measurable difference in the contrast or latitude of the 3 films, and the skin dose varied exactly as the mas. required to produce comparably exposed films.

There was a very significant difference in the resolution measured. Non-screen film produced appreciably less resolution than either type AA or type M. This was readily noticeable on the roentgenograms of both patients and specimens. No measurable difference in resolution between type AA and type M films was found. It is believed that resolution is limited by factors such as focal spot size, kv., and target skin distance, rather than by the grain when the grain becomes as small as that of type AA.

Resolution tests were made on Kodak Blue Brand with a single screen, DuPont Commercial-S with a single screen, and Ansco Hi-Speed with no screens. Resolution (as defined for mammography) was markedly inferior to that of the industrial films, and was not considered adequate for mammography. In consequence, further tests of these 3 films were not made. Ansco Super-Ray A and Super-Ray B, as well as Du Pont 506 and 510, were checked sufficiently to determine that they are approximately equivalent in most pertinent characteristics to Kodak AA and M, respectively.

The dependence of resolution upon geometric factors and photon energy (as it affects absorption coefficients) has been discussed by Rossman,[12] with results of tests on type M film.

A limited study was made of Polaroid film which is of some interest because it is extremely fast, and, by its use, mas. and consequently skin dose can be significantly reduced. However, the resolution and contrast of Polaroid film were so inferior as to

Table VI

EFFECT OF MILLIAMPERE SECONDS

Milliampere seconds	480	960	1,920
Reference optical density	1.0	2.0	4.0
Skin dose (rads)	3	6	12
Atomic number contrast	0.75	1.5	3.0
Thickness dependence	22	22	22
Resolution	56+	64	unreadable
Cooling time (min.)	0.4	0.9	2.1

lead us to the conclusion that it was not presently practical for use in mammography. Type AA film, whose resolution in mammographic application is approximately equal to that of type M and which has about 3 times the speed, is considered to be the best choice for mammography at this time.

Film development. The mas. required may be further reduced by increasing the time of development. Table v shows that increasing the time from 5 to 10 minutes permits a 40 per cent reduction in mas. for type M film and a 20 per cent reduction for type AA with no significant loss of quality.

Beryllium window tube. Preliminary tests of a beryllium window roentgen-ray tube designed with mammography in mind were carried out. Results indicated that the additional output offered by a beryllium window was largely unusable, consisting mostly of roentgen rays too soft to penetrate the breast and expose the film. The skin dose with unfiltered beryllium window radiation can amount to several hundred roentgens per film. Added filtration roughly equivalent to the inherent filtration of a glass tube is therefore mandatory in the 30 kvp. region. Aside from the window material, a feature of the particular tube tested was close spacing of cathode and target to reduce electron space-charge effects. This required that the tube could not be used above 60 kvp. It was also noted that anode heat storage was substantially below that of the conventional glass tube used for the other tests here reported.

At the present time, beryllium window tubes seem to offer no substantial advantage over glass tubes for mammography. If very fast films such as Polaroid are developed with sufficient resolution and contrast, it is possible that beryllium window tubes may then show considerable advantage. Measurements are being made with other available mammographic units, and the results will be published subsequently.

Studies on the use of a liquid bolus were carried out. Mineral oil was found to be an effective material. Other substances have been used.[1] The need for this special technique was found to be very small in actual practice.

MEMORIAL HOSPITAL TECHNIQUE

The technique in use for mammography in the Department of Diagnostic Radiology of the Memorial Hospital is given in Table VII. Exposures and kilovoltage are selected to give optimal contrast and resolution consistent with tube safety. Ample clinical experience has now been gained with this technique and it has been possible to compare the films so obtained with those of

TABLE VII

MEMORIAL HOSPITAL TECHNIQUE

Thickness of breast (cm.)	Kvp. and skin dose for density of breast					
	Low		Medium		High	
	Kvp.	r	Kvp.	r	Kvp.	r
4	29	1.6	30	1.8	31	2.0
5	30	1.8	31	2.0	32	2.2
6	31	2.0	32	2.2	33	2.4
7	32	2.2	33	2.4	34	2.6
8	33	2.4	34	2.6	35	2.9
9	34	2.6	35	2.9	36	3.1
10	35	2.9	36	3.1	37	3.3

Target skin distance, 28 to 32 inches; film, Kodak AA or Ansco Super-Ray A; milliampere seconds, 600 (may vary depending on density of film desired); added filter, ½ mm. Al. (Skin dose figures are for 600 mas.)

other techniques. The total experience has given confirmation of the general superiority of this technique.

Developing the exposure table. A simple rule of thumb may be used to determine exposures: each centimeter increase in breast thickness requires an increase of 1 kvp. For fatty breasts, 1 kvp. may be deducted from the standard value; for firm breasts, 1 kvp. may be added; for very firm breasts, add 2 kvp. The absolute kvp. values of Table VII will not apply to other roentgen-ray machines, due to differences in inherent filtration, wave form, etc. A base kvp. must be established by trial and error for each roentgen-ray unit once an mas. setting has been determined by tube loading and exposure time considerations. Having determined the base kvp. for a breast of known thickness, the "1 kvp. per 1 cm. change in thickness" rule of thumb may be followed, enabling each radiologist to develop his own complete exposure table.

CLINICAL CONSIDERATIONS

This project in its clinical application has been informative in several important technical aspects. A practical as well as an excellent mammographic technique is clearly mandatory and the entire breast must be satisfactorily visualized, for a lesion not on the film cannot be detected. Two projections, craniocaudad and lateral oblique are used routinely with an additional axillary or tail-of-the-breast view when indicated. In the lateral oblique projection, the film must be deeply placed against the chest wall and there must be close contact between the entire breast and the film. A cone centered to the mid-point of the breast is used with a target skin distance just great enough to include the entire breast without cutting any portion of it. Compression is not employed regularly. The shorter exposure time with AA film provides a distinct advantage over M film as respiratory motion is more easily avoided. All exposures are made in expiration.

The breast need not be examined directly by the radiologist prior to roentgenography but sometimes information concerning the clinical findings may be important in planning full coverage of areas in question. The radiologist should examine the mammograms before the patient leaves so that special studies can be made if needed. If this is not done, there will inevitably be a number of recalls. All mammograms must be read with the aid of magnification and a bright light beam.

Experience in interpretation is, of course, essential, for the complexities of detection and diagnosis are indeed manifold. Localization and removal of small lesions for biopsy may be frustrating to the surgeon and routine gross pathologic examination can easily bypass a small lesion that roentgenographic methods can now detect. A pathologic diagnosis made by frozen section not infrequently may be altered when the formal sections are studied. Close cooperation between the radiologist, surgeon, and pathologist is often necessary. Experience with carcinoma *in situ* has demonstrated the need for this cooperation.[14]

Errors in detection and diagnosis by the radiologist may not be revealed unless all patients are followed for a sufficiently long period and initial mammograms are carefully compared with the latest ones. In other words, a breast called negative for cancer roentgenologically is not necessarily so in fact, and statistics employing such a basis are open to question. The tendency to overdiagnose cancer in order to establish a near perfect accuracy in the detection of this disease leads to excessive use of biopsy, creates a serious inaccuracy in the recognition of benign conditions, and weakens the confidence of the referring physician in the method.

SUMMARY

A series of tests has been carried out to determine an optimal technique for mammography. The technique developed has been tested in over 2,000 examinations. Essential points are the use of Kodak AA film or its commercial equivalent; fixed

milliampere seconds and a relatively fixed target skin distance; and a variable peak kilovoltage determined by breast thickness and composition. Variations in roentgen-ray apparatus prohibit confident direct transfer of kvp. and mas. values; however, variations from a trial-and-error-determined base kvp., determined by a "1 kvp. per 1 cm. change in thickness" rule, may be transferred. Clinical experience with the technique and other technical considerations are discussed.

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Operative specimens and patients were provided by members of the Breast Service, Dr. Joseph H. Farrow, Chief.

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A NEW CONTOUR CASSETTE FOR MAMMOGRAPHIC ROENTGENOGRAPHY*

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WITH increased general usage of mammography (roentgenography of the breast), the need for a more satisfactory cassette for study of the breast in the craniocaudad projection became apparent. With the usual craniocaudad projection made on a conventional rectangular film, large segments of both the medial and the lateral portions of the breast near the chest wall cannot be included on the film. For this reason a cassette has been designed which conforms to the contour of the chest wall and permits demonstration of a much greater portion of the breast in this view (Fig. 1).

A rectangular cardboard cassette (film holder), 10 by 12 inches, was obtained and cut to the general contour of the anterior chest wall, as shown in Figure 2. In the dark room, the following procedure is used to cut the unexposed film to fit the contour cassette. The cassette is placed on top of the 10 by 12 inch roentgenographic film and the outline of the contour edge is traced on the film with a blue wax pencil. The film is then cut with a pair of scissors. This blue line can be seen easily with the illumination provided by the normal safe light in the darkroom. A number of films can be cut and stored in the regular darkroom film bin for future use.

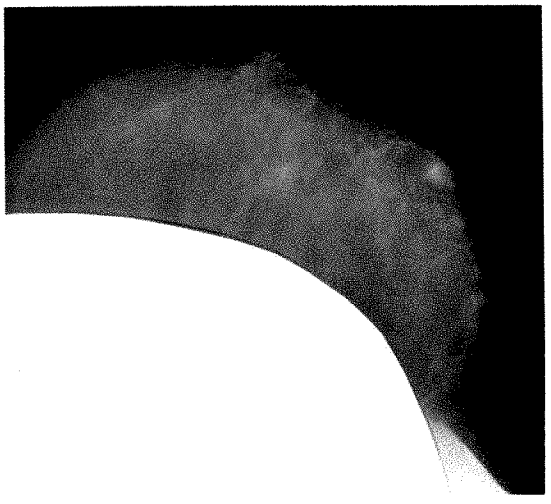


FIG. 1. Craniocaudal roentgenogram of the right breast. The contour film provides for examination of considerably more mammary tissue than does the rectangular film.

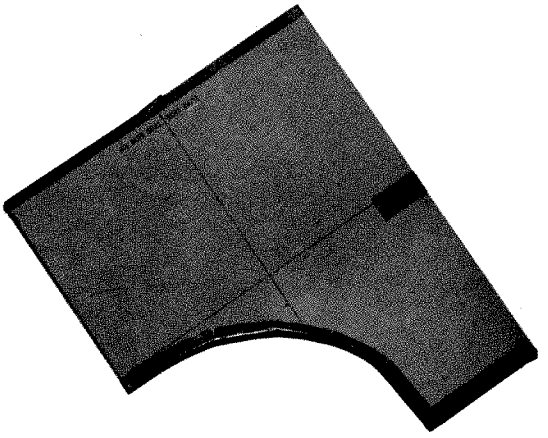


FIG. 2. The contour cassette.

The contour film permits examination of a greater portion of both the medial and the lateral aspects of the breast in the craniocaudad projection than is possible on the routine film. It also can be used for spot checking a suspicious lesion evident on the routine roentgenogram.

Because three corners of the film are left undisturbed, it can be fastened to the hangers which are used for roentgenographic film processing and thus processed in the usual manner.

In use, the contour cassette is supported by a piece of masonite board about 6 inches longer than the contour cassette but with

* This work was carried out under the supervision of David M. Witten, M.D.

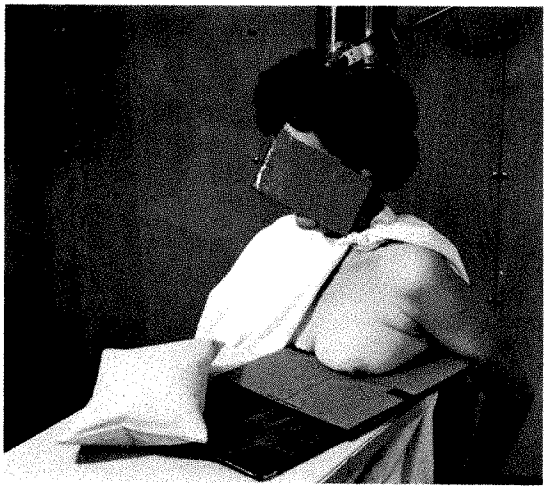


Fig. 3. A model demonstration of a contour cassette and film used to obtain a craniocaudal view of the breast. The cassette is placed in apposition to the chest wall beneath the left breast.

an edge cut to conform to the contour of the cassette. The board is placed with the shaped edge extending over the edge of the table next to the patient; a sandbag is set on the opposite end to hold the board in position.

The patient sits as erectly as possible in a chair next to a table that has an adjustable top. The head is tilted slightly away from the side to be roentgenographed. The contour cassette is placed under the breast to be roentgenographed with the contour portion in close contact with the chest wall. The roentgenographic tube is centered directly above the cassette so that the central beam passes through the center of the breast, as shows in Figure 3.

SUMMARY

A simple contour cassette has been developed for mammography. Its use permits a greater portion of the breast to be examined roentgenologically in the craniocaudad projection than is possible on the routine rectangular craniocaudad roentgenogram. The contour cassette is simple to use and the roentgenographic film can be cut and processed with ease. The use of a contour film is a valuable aid to mammographic roentgenography.

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THERMOGRAPHY OF THE BREAST*

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ROENTGENOLOGISTS as a result of their training, readily comprehend the meaning of thermoradiography or thermography, a new diagnostic discipline concerned with the infrared portion of the same electromagnetic spectrum which includes roentgen rays. Thermography is a term suggested to describe the art of obtaining an image reproduction of the infrared radiation spontaneously emitted by an object under study. Radiation of energy from the human body requires no medium of transfer and does not have to be stimulated by irradiation of any type. Infrared rays travel in straight lines at the speed of roentgen rays or light, falling off in intensity as the square of the distance; and can be focused, reflected, refracted and recorded by appropriate means on photographic film, electrochemical paper or on television monitors.

The spectrum of infrared rays, in the far infrared region, emitted from the surface of the human body depends on the absolute temperature and the character of the surface, namely the skin. Since the skin is almost a perfect absorber of infrared radiation in the portion of the spectrum used for thermography, it may be regarded to a first approximation as a black body and so a perfect emitter.

Infrared detectors are now available to measure surface temperatures at great distances. Modern techniques, for example, have made it possible to detect the heat of a match at 20 miles with a response time of the order of microseconds. Scanners are available which will record heat images or thermograms on photographic film or electrochemical paper. Such a scanner, originally designed for military purposes, was recently described by Barnes.[1] This thermograph has been in daily use in this institution for over a year and has quite recently been replaced by a new model, the Barnes Thermograph M-1. Two of these instruments together with two portable infrared thermometers and other ancillary equipment now form the basic instrumentation in a new Division of Thermography of the Department of Radiology.

METHOD AND RESULTS

To eliminate spurious results due to changes in ambient temperature, the effect of clothing, exercise, perspiration, etc., it is necessary for the skin to be normalized. Patients recline in a room at 70–76° F. for about 10 minutes and when the breasts are examined, the arms are extended to avoid cross radiation of the axillae. The emitted infrared radiation is reflected and focused into the camera by an overhanging front-silvered mirror angled at 45°. Depending on how large a field is scanned, exposures are completed in 4 to 12 minutes and the thermogram, printed on Polaroid film, is available 20 seconds later. A scale of gray half-tones is imprinted on each film ranging from white to black in eight steps and can be interpreted to span from 1° C. to 10° C. depending on the contrast setting employed for the particular lesion under study.

All patients having roentgenographic examinations of their breasts have been routinely thermographed. To date, some 750 patients have been studied of whom about 150 have been operated on, including 40 cases of breast cancer.

The normal breast in the thermogram can be recognized by its size, shape and the heat pattern of the overlying skin. The

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nipple is identified by a black spot produced on the film by a small circular tab of aluminum foil (a poor emitter) placed on the nipple before scanning. Cool areas of the skin are portrayed in darker shades while warm areas appear in lighter shades. Temperature differences can be calculated from the gray scale as illustrated in Figure 1, *A*, *B* and *C* which shows a case of an infiltrating duct carcinoma.

Tissues beneath the skin vary in their metabolic activity and blood supply; and of course, the skin itself has an exceedingly rich blood supply which varies widely under different conditions of health, disease and medication. The various sources of heat combine to create localized temperature differences on the skin which can be thermographed and correlated with various physiologic and pathologic conditions.

Malignant lesions of the breast can usually be spotted on thermograms by the bright area over a patch of skin with temperature differences varying from 1° C. to 7° C.[2,6] Not only can the thermographic findings confirm clinical and radiologic opinions, but they can also help formulate correct interpretations under otherwise questionable circumstances. For instance, in the following cases, thermography was the only diagnostic approach that indicated the pathology.

REPORT OF CASES

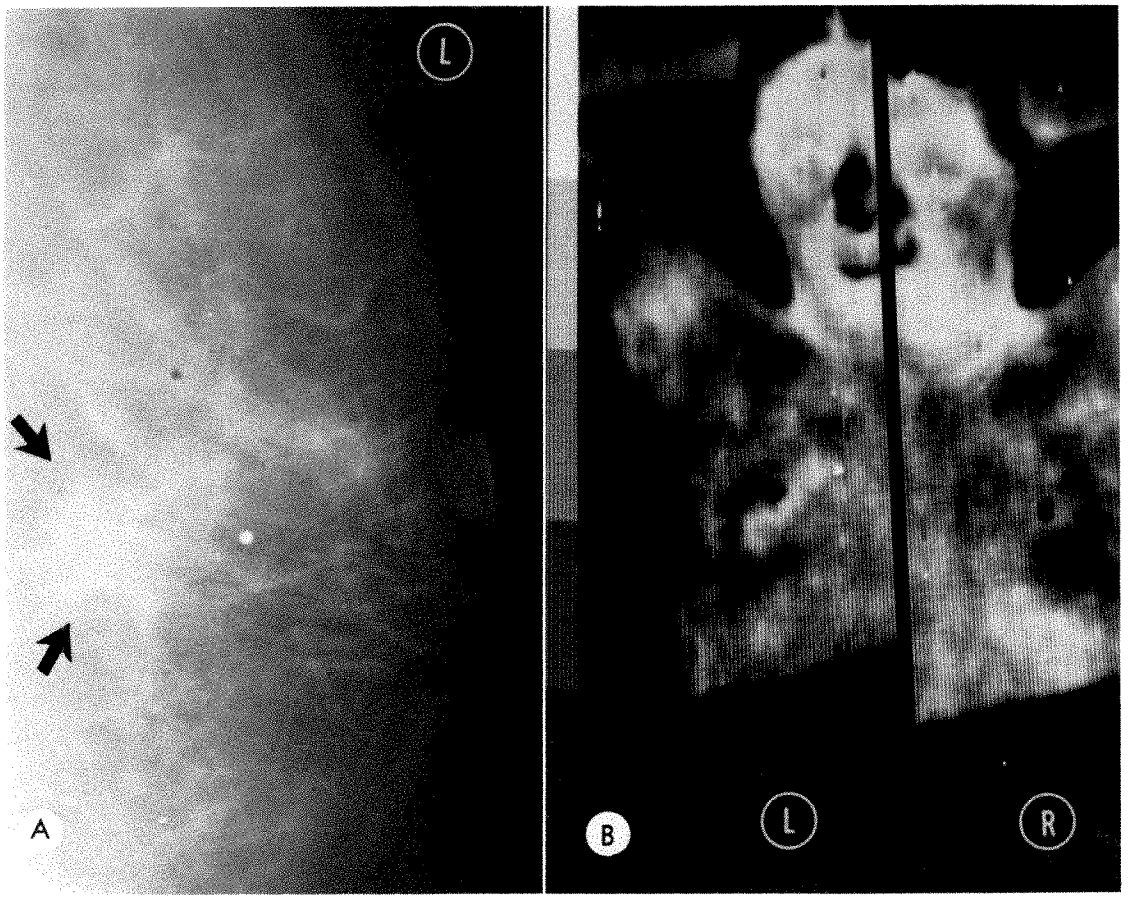
CASE I (C-6857). Shortly after a cyst was removed from the right breast of this 38 year old patient, she noted another mass in the tail of the breast. After a delay of 1 year, she finally consulted her physician who confirmed the presence of a firm, irregular, tender mass adjacent to the scar which he regarded as another cyst. Roentgenograms revealed what was reported as either accessory breast tissue or an area of mazoplasia cystica. Thermography disclosed increased skin temperature variations over both breasts, but nowhere exceeding 1° C. except over the area in question where the difference was 1.4° C. On this basis, a biopsy was urged and at the time of the operation the pathologist reported from the frozen section cystic disease. Later, after study of the permanent sections, he changed his diagnosis to lobular carcinoma. Here then, we were confronted with a case in which the clinician, surgeon, radiologist and pathologist had difficulty in arriving at a diagnosis, whereas the thermographic evidence indicative of malignancy was proved correct (Fig. 2, *A* and *B*).

CASE II (C-7153). After discovery of a lump in the left breast, this patient consulted her physician who suggested a re-examination because he did not believe the mass to be dominant or associated with findings suggestive of malignancy. Re-examination 6 months later revealed no changes. Mastography was then done and disclosed a sharply circumscribed mass which resembled a benign process. Fortunately, biopsy was suggested because of the possibility of an unrecognized malignancy and when thermography revealed 1.3° C. skin temperature elevation over the lesion, which strongly suggested a malignant process, biopsy was done without delay. The pathologist reported an infiltrative duct carcinoma with no evidence of metastasis to the axillary lymph nodes. This case again demonstrated the value of thermography in the presence of equivocal clinical and roentgenographic findings (Fig. 3, *A* and *B*).

CASE III (C-6855). During a routine examination, this patient's physician palpated a lump in the right breast. Roentgen studies failed to identify any changes suggestive of malignancy, but thermography revealed 2.3° C. elevation of skin temperature over the lesion compared with adjacent areas. Because of these findings, a surgical consultation was obtained, but again no dominant mass or changes in the breast to suggest malignancy were found. Nevertheless the surgeon was prevailed upon to do a biopsy which disclosed the presence of a scirrhous carcinoma about 1.0 cm. in diameter with no involvement of the axillary lymph nodes. This case again illustrates the value of thermography in arriving at a correct diagnosis of malignancy in the absence of clinical or roentgenographic evidence (Fig. 4, *A* and *B*).

COMMENT

Benign diseases of the breast are reflected by various mosaic patterns probably related in part to the distribution of subcutaneous veins, in part to the character of the



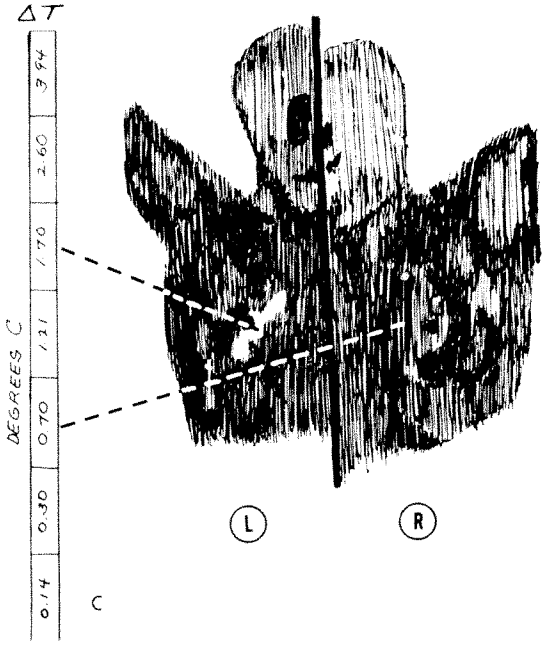


FIG. 1. (*A*) Roentgenogram of infiltrating duct carcinoma of left breast located approximately 3 cm. below skin surface in mid-line just below level of nipple. (*B*) In the thermogram of both breasts, a rectangular light gray zone due to increased infrared emission runs upward and inward below the left nipple. (Cross irradiation in the right axilla because the arm was not completely extended accounts for the temperature elevation in this area.) (*C*) Diagrammatic illustration of *B*. When the light zone of the left breast is compared with a similar area of the opposite breast with reference to the standardized gray scale, there is a temperature difference of 1° C. Malignancy is thus suspected insofar as most benign processes are associated with lower skin temperature elevations. Fat is a poor conductor of infrared rays, yet in this case enough conduction occurred through a layer of more than 3 cm. to be of diagnostic value.

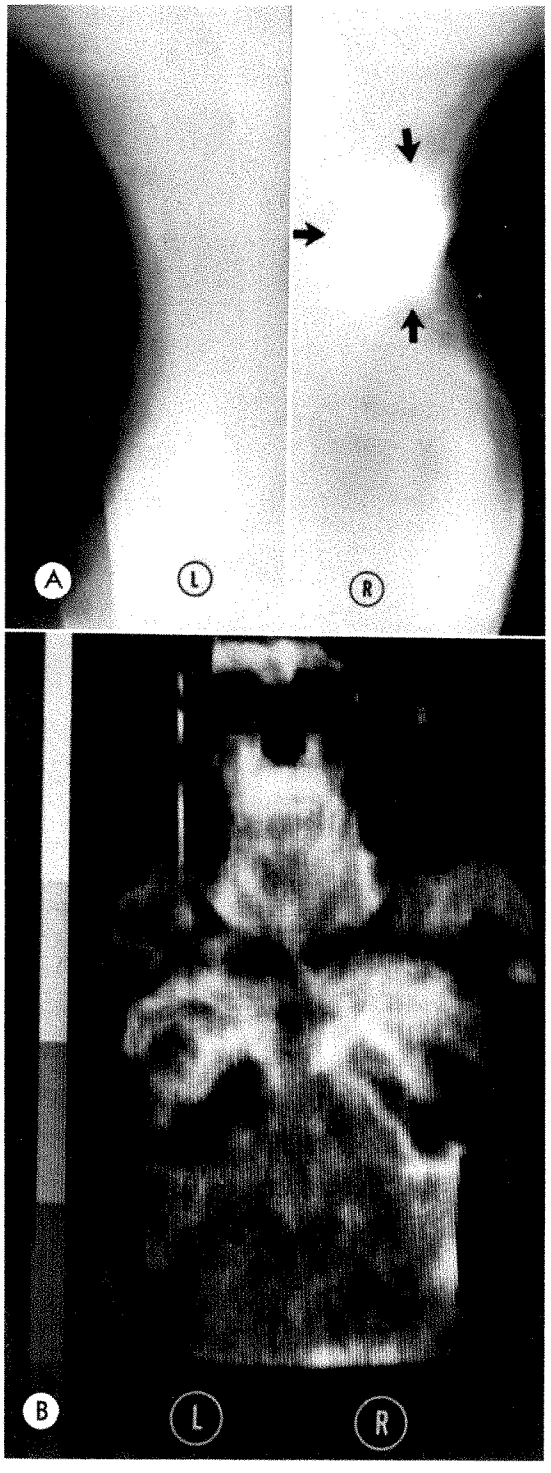


Fig. 2. Case I. (*A*) Mastograms show changes in the tail of the right breast near the axilla, reported as either due to accessory mammary tissue or mazoplasia cystica. (*B*) Thermogram reveals a mosaic of warm patterns over both breasts, but the difference of skin temperature is nowhere in excess of 1.0° C. except over the area of the lesion where the difference was 1.4° C. For this reason, the probability of malignancy was suggested and finally confirmed as lobular carcinoma by the pathologist.

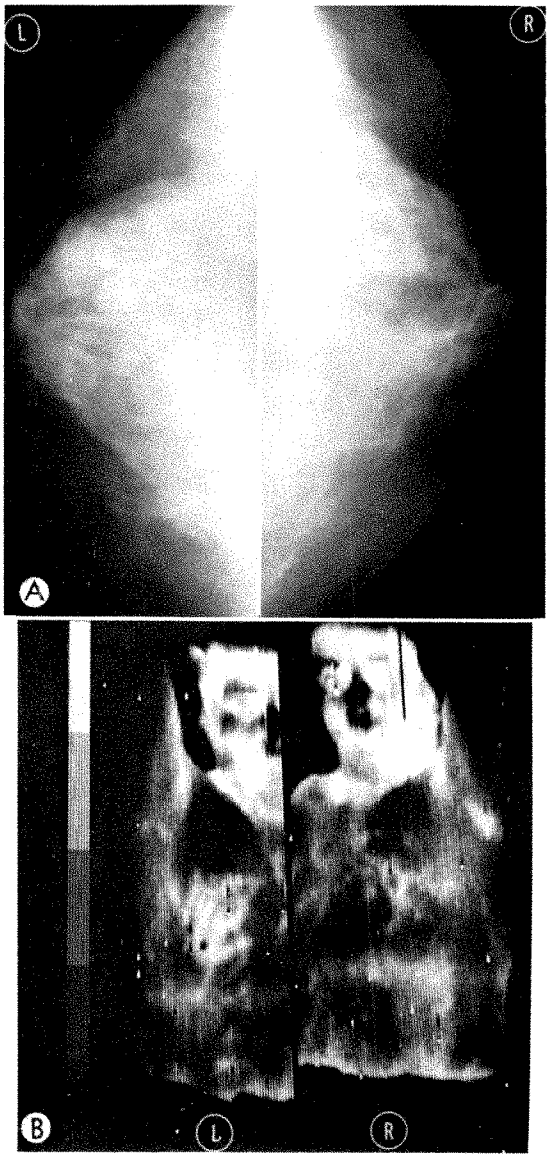


Fig. 3. Case II. (*A*) Mastograms show a compact, circumscribed mass in the left breast suggestive of a benign process. Biopsy was advised only because it was realized that the pattern did not preclude the possibility of malignancy. (*B*) Thermogram reveals increased skin temperature over much of the left breast, but in one area, the elevation reached 1.3° C., suggesting a malignant process. An infiltrating duct carcinoma was found without evidence of metastasis to the axillary lymph nodes.

fat content of the breast and, finally, in part to the metabolic activity of the benign process. Areas of slightly elevated temperature which frequently appear within the darker cool skin surfaces of the average breast seem due to the increased heat conducted to the skin from subcutaneous veins. The broader expanses of darker shades of gray over the breast are usually due to the fat in the breast so that the less fat, the higher the temperature, the greater is the emission and the lighter are the shades. When computations of the skin temperature differences are made with reference to the standardized gray scale on each thermogram, seldom are they found to be more than 1° C. over benign processes. In certain cases temperature differences greater than 1° C. have been recorded in cases where the only lesions found by the pathologist proved to be certain types of fibroadenoma or inflamed cysts. Interpretation by thermography of breast disease, therefore, cannot be made out of context; other diagnostic findings, including those of mastography, should also receive due consideration. At the moment, we do not understand why some benign processes should cause skin temperature elevations of 1° C. or more, but we suspect that increased cellular proliferation and metabolic activity may be responsible. Just as with the increased cellular proliferation of the breast which occurs during the later stages of pregnancy when there is increased elevation of skin temperature, so in active cellular proliferations and infiltrations which occur during certain phases of mazoplasia, fibroadenoma or inflammation, similar skin temperature elevations may also occur. In Figure 5, a case of pregnancy is illustrated. The *generalized* skin temperature elevation over both breasts, therefore, may be similar to the *localized* skin temperature elevation in benign conditions as shown in the breasts of Figure 6, *A* and *B*. After sufficient studies have been accumulated, some other level of skin temperature differences than 1° C. may be found more appropriate for distinguishing malignant from benign conditions in the breast; but we would venture to say

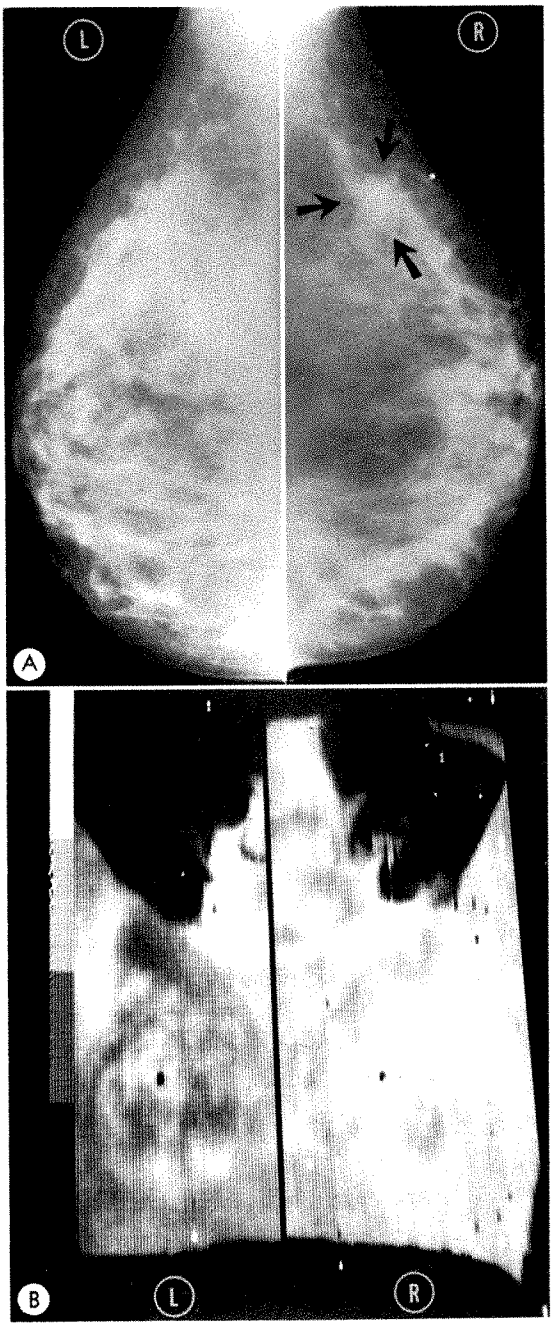


Fig. 4. Case III. (*A*) Mastograms show distorted trabecular pattern in the upper right breast not recognized as due to malignancy. (*B*) Thermogram reveals diffuse increased skin temperature elevation over the entire right breast, but over the lesion the elevation was 2.3° C., thus suggesting the presence of malignancy. A small scirrhous carcinoma was found without axillary lymph node metastasis.

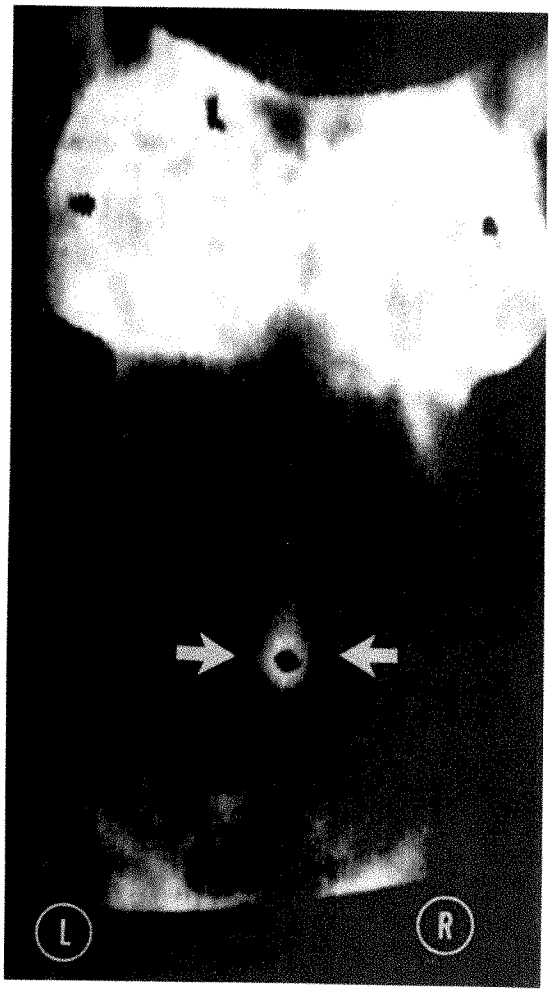


Fig. 5. Thermogram of a patient in the eighth month of pregnancy showing increased heat emissivity ($\Delta T = 1.4°$ C.) accounting for the light gray tones over both breasts. (Cross irradiation in the navel [arrows] accounts for localized elevated skin temperature.) Nipples and center of navel are marked with aluminum disks.

that no matter what arbitrary level is found from statistical analysis of the data to be most appropriate, exceptions will still be found requiring interpretation of thermograms in the context of all other diagnostic data.

DISCUSSION

The infrared portion of the electromagnetic spectrum which was discovered by Herschel in 1800 has come into general prominence only during the last 3 decades. Every object in the world constantly emits infrared radiation as a function of its absolute temperature. The earth itself is exposed to and absorbs enormous quantities of heat energy and yet it remains in thermal equilibrium with its surroundings at an average temperature of about 300° Kelvin or 80° F. It absorbs more than it emits on the side facing the sun and emits more than it absorbs on the other side. Every object, in attempting to maintain thermal equilibrium, is constantly either absorbing or emitting radiant energy with its line-of-sight neighbors. It follows that a good absorber must be an equally good emitter. The skin is a good absorber; therefore, it also is a good emitter and, within limits, helps humans maintain a correct body temperature.

The total infrared energy emitted or radiated by an object is a function of the fourth power of its absolute temperature. Since in the infrared, skin is almost a perfect absorber, man himself also emits invisible light which may be recorded on the thermogram in the delicate half-tone shadings quantitatively proportional to minute temperature differences which actually exist over the surface.

Before the present day clinical thermometer came into general use, about 270 years elapsed between Galileo's original observations and Wunderlich's extensive studies in 1865. Even now, we continue to record the normal body temperature at 37° C. or 98.6° F., whereas actually we are only measuring the oral temperature. The rectal temperature is higher than that of the mouth or the skin. The temperature of the skin varies in different areas, that of the foot normally being higher than that of the toes and that of the leg higher than that of the foot. Local elevation of temperatures in inflammatory or neoplastic processes often occurs without being detected by oral or rectal determinations. The actual recording of temperatures with the clinical thermometer or a thermocouple necessitates actual contact with

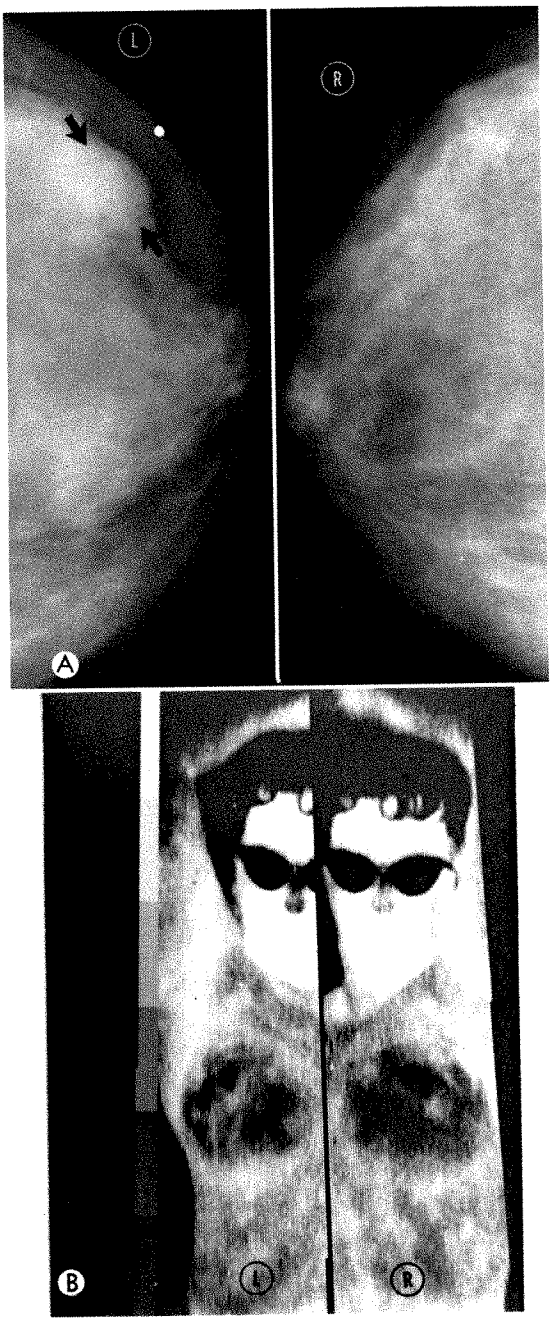


Fig. 6. (*A*) Mastograms reveal predominant mazoplasia cystica in left breast and adenosis in right breast. (*B*) Thermogram of breasts. Greater temperature differences are visible over the left than over the right breast, but none exceeds 0.5° C. During a routine physical examination of this 47 year old patient, a mass was palpable in the upper outer quadrant of the left breast. A biopsy was done on both breasts because, in addition to the mass in the left breast, another mass was found in the right breast a day before the operation. The pathologist reported cystic disease of the left breast and fibroadenoma of the right breast.

tissues, which in itself interferes with the accuracy of recording. Heat sensing instruments like that used in the present work overcome this handicap since no actual contact is made with the body.

Lawson[4] was the first to point out the virtues of thermography in 1957. His subsequent reports[5] and those of Williams, Williams and Handley[6] further advanced acquaintanceship with this discipline. Williams *et al.*, in a series of 57 cases of breast cancer, were able to correctly establish the diagnosis in 54 by the use of temperature mapping over the breasts using a hand-held thermopile. In a private communication, they report extension of this series to 100 cases of carcinoma, 95 of which were correctly diagnosed. Their experience with thermographic scanning using electrochemical paper was limited, but suggested a promising future.[6]

As more data are accumulated by various investigators in this new field of diagnosis, breast thermograms will be more extensively appraised and analyzed from a nosologic point of view.[3] It must be realized that thermography is in its earliest stages of development and to appraise its value merely on its accomplishments so far is to take a short-sighted view of its potentialities. The rate of improvement in thermographic apparatus and its technical application depends on enthusiastic trial by many experimenters on a broad front. It is hoped that in contrast to the slow appreciation of the potentialities of the infrared portion of the electromagnetic spectrum by physical scientists preceding the last 3 decades, medical investigators will be more alert in accepting the alluring challenges now confronting them in this medium. Research

and development associated with military and space programs already have led to the use of apparatus capable of rapid and precise recording of infrared radiation. Not to take advantage of these advances will result only in delaying the application of a significant contribution to medical science.

SUMMARY

Thermograms are half-tone pictures obtained with scanning apparatus of infrared radiation spontaneously emanating from the skin. Since the skin temperature is greater over diseased processes, more infrared is radiated and lighter half-tones representing inflammatory or malignant disease are distinguished from the darker half-tones which represent benign processes or areas of normal tissue. Thermograms of breasts involved in both benign and malignant disease are presented in this report. The results suggest that thermography may materially advance the diagnosis of breast diseases.

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Sixty-fifth Annual Meeting: Radisson Hotel, Minneapolis, Minn., Sept. 29–Oct. 2, 1964.

* Deceased.

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Representative on the Board of Chancellors of the American College of Radiology: Milford D. Schulz.

Forty-sixth Annual Meeting: Greenbrier Hotel, White Sulphur Springs, W. Va., April 13–16, 1964.

EDITORIALS

RELIABILITY OF THE ROENTGENOLOGIST AS A GASTROINTESTINAL CONSULTANT

THE reliability of roentgenologic examinations in the diagnosis of diseases of the gastrointestinal tract is of such fundamental importance to our specialty that it is surprising so few analyses of it are available. All roentgenologists will profit from careful study of the extensive, conscientious appraisal recently undertaken by Cooley.[1,2]

Earlier studies[3,4] are also available for those who may seriously wonder what our efforts have accomplished in the field of medical diagnosis and what factors have limited our techniques. In 1950 Sosman[3] presented an extremely interesting philosophical discussion of the specificity and reliability of roentgenographic diagnosis. In discussing gastroenterology, he stated, "In no other anatomic field is the physician or surgeon so heavily dependent upon the roentgenographic method for so many of his decisions." He pointed out that a proper gastrointestinal examination "requires the greatest time, skill, patience, effort and judgment of any roentgenographic examinations, with the sole exception of the highly specialized fields. The diagnostic dividends, moreover, are in direct proportion to the training and skill of the examiner." He stressed the desirability of intimate cooperation of the patient's physician and roentgenologist in sharing their suspicions, facts, and experience. He contrasted gastrointestinal roentgenology, in which the major problem is demonstration or detection of a lesion, with the problem in thoracic roentgenology, in which most lesions are easily demonstrated but difficult to identify specifically, because of the numerous causative possibilities.

In evaluation of the differential diagnostic possibilities, Sosman stressed the importance of considering the relative frequency of any given lesion as modified by the patient's age, sex, and race. He also advised the examiner to help the referring physician as much as possible by indicating whether his diagnostic deduction was certain, highly probable, probable, possible, or a normal variation. When a radiologist performs in this fashion, his reliability depends on a broad knowledge of medicine and radiology, as well as on technical finesse in demonstrating the presence of disease. Balanced judgment is required. As competence is repeatedly demonstrated in daily diagnostic problems, the referring physicians come to rely increasingly upon the expressed opinions of the examining radiologist. This kind of reputation can be earned only by years of careful work and studious recognition of more than only the obvious diagnostic clues in the flow of daily examinations.

The roentgenologist must know the limitations of his method if he is to achieve a reputation for reliability. In 1949, Rigler[4] pointed out that the objectivity and visual appeal of the roentgenologic diagnosis inspired in physicians as well as laymen a confidence and reliance that was not always merited by its accomplishments. He advised us to think of "the term limitations in its broad sense, that is, as meaning the maximal as well as the minimal possibili-

[1] Cooley, R. N. The diagnostic accuracy of upper gastrointestinal radiologic studies. *Am. J. M. Sc.*, 1961, *242*, 628–650.

[2] Cooley, R. N. The diagnostic accuracy of radiologic studies of the biliary tract, small intestine and colon. *Am. J. M. Sc.*, 1963, *246*, 610–638.

[3] Sosman, M. C. The specificity and reliability of roentgenographic diagnosis (The Shattuck lecture). *New England J. Med.*, 1950, *242*, 849–855.

[4] Rigler, L. G. The possibilities and limitations of roentgen diagnosis (Pancoast lecture). Am. J. Roentgenol. & Rad. Therapy, 1949, *61*, 743–761.

ties." If we have knowledge of, and stay within these boundaries of our method, our diagnostic reliability will certainly improve.

In addition to the inherent limitations of our method of examination, our diagnostic reliability is subject to another modifying influence because radiologists share the usual failings to which all human beings are subject. Even in cases in which definite abnormalities may be identifiable in roentgenograms, the observations are subject to individual interpretation and the radiologist must remain aware of the natural element of fallibility and variability of judgment that human beings exhibit. In the words of Garland,[5] "In nearly every activity which can be tested, it has been repeatedly demonstrated that humans, even experts in a given field, exhibit enormous variations in their ability to be consistent with themselves and others equally competent." He pointed out that the medical profession, despite long study and training, cannot expect to be free of this foible. Any radiologist who follows his cases or has an opportunity to review or re-examine former patients at a later date must occasionally be surprised by some of his previously rendered opinions or his errors of oversight.

In his review of the results obtained in diagnosing many specific lesions, Cooley[1] listed a number of general faults and omissions in many of the reports in which the accuracy of roentgenologic diagnosis is discussed. These include the limited data on false negatives (incorrectly diagnosed as normal) and false positives (incorrectly diagnosed as abnormal), lack of follow-up data on negative cases, incomplete data on total number of examinations from which positive cases were selected, analyses of retrospective type that inaccurately portray the situation at the time of critical diagnostic decision, uncertainty about completeness of medical records, and failure to differentiate what some special group or interested individual may accomplish as opposed to the average performance of the average radiologist. Anyone who has participated in a special study of some problem or prepared a paper for publication realizes that these criticisms are valid and that they prevent many reports from being ideal studies.

How to remedy these faults is a more difficult problem. In some types of study they are insuperable. Other reports might be improved if these weaknesses are kept in mind. Despite the many hiatuses in our knowledge, Cooley was able to collect considerable information which may act as guide lines for our consideration of future work. There is little doubt that our reliability will be sharply improved if we become more aware of our major errors of inaccuracy and conscientiously try to improve our techniques, widen our horizon of interest, and deepen our channel of knowledge.

Seymour Fiske Ochsner, M.D.
Ochsner Clinic
New Orleans 21, Louisiana

[5] Garland, L. H. On scientific evaluation of diagnostic procedures. *Radiology*, 1949, *52*, 309–328.



JOSHUA CLIFTON DICKINSON
1885-1964

WITH the death of "Doctor Dick," the era of the pioneers in Florida Radiology came to an end. Joshua Clifton Dickinson died on January 9, 1964 of hemorrhage from, and perforation of, an acute esophagogastric ulcer.

Dr. Dickinson was born on January 29, 1885, on a stock farm near Bellefontaine, Ohio. He received his elementary and high school education in this Ohio community and then went directly to medical school, as was the custom in those days. He received

his degree of Doctor of Medicine from the Cincinnati Eclectic Medical School in 1907. Immediately after graduation, he journeyed out to Los Angeles with the firm determination to practice general medicine in that community, but after a short time decided he did not like California and came directly to Tampa, arriving in the fall of 1908.

Opening an office in what is now Tampa's Ybor City, Dr. Dickinson practiced general medicine which, in those days, included obstetrics and minor surgery. It was during this period of his life that his joy and pride in being a doctor developed, a feeling which he carried with him throughout his entire professional career; Dr. Dickinson was always, first and foremost, a physician. Although his original intent had been to eventually specialize in urology, the small general hospital of the community possessed the only X-ray equipment in the area, and it was here that his interest in radiology was born and began its development. Over the ensuing years, through his own hard work, study, and personal application, aided and abetted by short courses at the Johns Hopkins Hospital and the University of Illinois, he became one of the outstanding radiologists in the south, if not in the nation.

He felt quite strongly his obligations to organized medicine and radiology, maintaining an interest in organizational work throughout his life. He served as secretary and as president of the Hillsborough County Medical Society in 1923. In the fall of 1931 he, with other radiologists in the state, organized the Florida Radiological Society. During its early years he served on the Board of Censors, later both as its secretary and as its president. He maintained his membership and his interest in the Florida State Medical Association, the Southern Medical Association, and the Radiological Society of North America. He served as chairman of the Section of Radiology of the American Medical Association, as a member of the Board of Chancellors of the American College of Radiology, and as a member of the Executive Council of the American Roentgen Ray Society. In 1954, he served as president of the American Roentgen Ray Society, an honor which he always felt represented the apogee of his career.

Dr. and Mrs. Dickinson were not prone to enter into society in the grand manner, preferring to entertain their many friends in the comfort and intimacy of their own home. However, he was a charter member of the Palma Ceia Golf and Country Club, the Tampa Yacht and Country Club, the First Presbyterian Church of Tampa, Ye Mystic Krewe of Gasparilla, and the Bellefontaine (Ohio) Lodge, A & FM. His hobbies were few, his overwhelming interest being in the practice of medicine. At one period in his life he enjoyed golf. Later on it was duck shooting and fishing. Still later, after retirement, he derived a great deal of pleasure fulfilling the role of Oldest Member at the golf club.

On December 20, 1913, Dr. Dickinson married Miss Mary Louise Martin, hers being one of the old families of Virginia. Their one child is Joshua Clifton Dickinson, Jr., Associate Professor of Biology, University of Florida, and Director and Curator of Ornithology, The Florida State Museum. In addition to Mrs. Dickinson, his son, and his daughter-in-law, Dr. Dickinson is survived by a brother, E. W. Dickinson of Tampa; a sister, Mrs. Cleo Bushong of West Mansfield, Ohio; three grandchildren and two great grandchildren.

With the passing of Dr. Dickinson, those of us who have loved him dearly, and all of Radiology, have lost a wonderful friend and a wise counselor.

Charles M. Gray, M.D.

NEWS ITEMS

AMERICAN COLLEGE OF RADIOLOGY

The Fortieth Annual Meeting of the American College of Radiology was held at the Ramada Inn, Tucson, Arizona, on February 5–8, 1964. The following officers were elected: *President*, Dr. David S. Carroll, Memphis, Tennessee; *Vice-President*, Dr. Richard H. Chamberlain, Philadelphia, Pennsylvania; *Secretary-Treasurer*, Dr. Fay H. Squire, Chicago, Illinois (re-elected); *Chairman, Board of Chancellors*, Dr. Wallace D. Buchanan, South Bend, Indiana. Drs. George Cooper, Jr., Charlottesville, Virginia, Joseph D. Calhoun, Little Rock, Arkansas, Homer V. Hartzell, Seattle, Washington, Karl C. Corley, Washington, D. C., and William L. Sloan, Vancouver, British Columbia, Canada, were elected to the Board of Chancellors.

The Convocation Ceremonies were held on February 7, 1964. At these ceremonies Dr. Theodore J. Wachowski, Wheaton, Illinois, President of the College, conferred the Degree of Fellow on 41 candidates, and the Degree of Associate Fellow on 1 candidate. The Degree of Honorary Fellow was bestowed on Dr. Med. Luigi Turano, Rome, Italy, President-Elect of the Eleventh International Congress of Radiology and *in absentia* on Dr. James Ralston Kennedy Paterson, C.B.E., M.C., M.D., F.R.C.S., Stenrieshill, Wamphray, Moffatt, Dumfriesshire, England.

At the same ceremonies Dr. Wachowski, assisted by the new President, Dr. Carroll, presented to Dr. Bernard Pierre Widmann, Philadelphia, Pennsylvania, the Gold Medal, the highest award of the American College of Radiology "for distinguished and extraordinary service to the College and to the profession for which it stands."

The Forty-First Annual Meeting of the College will be held at Bellvue Stratford Hotel, Philadelphia, Pennsylvania, February 10–13, 1965.



Dr. Bernard Pierre Widmann, Past President of the American Roentgen Ray Society, was honored on February 7, 1964 by the American College of Radiology with presentation of the Gold Medal, the highest award of the College "for distinguished and extraordinary service."

COURSE IN RADIOLOGY OF THE HEART

A 4 day course, sponsored by the Department of Radiology of the Indiana University School of Medicine, is planned primarily for the clinical radiologist and cardiologist but is open to all physicians having an interest in this subject. It consists of lectures, panel discussions, film interpretation conferences and exhibits arranged so as to correlate the technical, physiological, clinical and pathological aspects of radiology of

cardiovascular disease in the pediatric and adult fields.

The course will be held April 8–11, 1964 and will be presented by members of the faculty of Indiana University School of Medicine and a distinguished guest faculty from various parts of the country.

For further information write to: Director, Postgraduate Education, Indiana University School of Medicine, 1100 W. Michigan Street, Indianapolis, Indiana 46207.

REFRESHER COURSE IN DIAGNOSTIC ROENTGENOLOGY

The Sixth Annual Refresher Course in Diagnostic Roentgenology will be held May 25–29, 1964 by the Radiology Department of the University of Cincinnati College of Medicine under the direction of Dr. Benjamin Felson, Professor and Director of the department. The course will include, in addition to lectures and demonstrations, teaching methods employing audience participation. The course is open to radiologists and radiology residents.

Further information can be obtained by writing Dr. Jerome F. Wiot, Department of Radiology, Cincinnati General Hospital, Cincinnati 29, Ohio.

POSTGRADUATE COURSE IN PEDIATRIC RADIOLOGY

The Annual Postgraduate Course of Children's Hospital of the District of Columbia will be presented May 20, 21, 22, 1964 on the subject of Pediatric Radiology. The topics to be presented will include uroradiology, neuroradiology and radiologic aspects of malignancies, tuberculosis and other chest diseases, and gastrointestinal disorders. It will be highlighted by the Wall Memorial Lecture on Congenital Malformations by Dr. Josef Warkany. It is sponsored by Children's Hospital of the District of Columbia in conjunction with the faculties of the Georgetown University School of Medicine, Department of Pediatrics and George Washington University School of Medicine, Department of Pediatrics, and is to be held in the Wall Memorial Auditorium of Children's Hospital. The capacity of the auditorium makes advance registration essential.

For further information, contact Donald W. Delaney, M.D., Assistant Director for Education, Children's Hospital of the District of Columbia, 2125 13th Street, N.W., Washington 9, D. C.

SYMPOSIUM ON ACUTE LEUKEMIA

A group of clinical experts and researchers will discuss the growing problem of acute leukemia in a symposium sponsored jointly by the American Cancer Society and the National Cancer Institute at the New York Hilton Hotel on May 22, 1964. The meeting will open at 9:30 A.M. with Dr. Sidney Farber, Director of Research at Children's Cancer Research Foundation, Boston, and Professor of Pathology, Harvard Medical School, as Chairman.

This symposium has been planned as an experiment in medical communication. It is intended to bridge the gap between laboratory research and care of the leukemia patient. About 12 scientists will participate, covering the following subjects: epidemiology, etiology, natural history and diagnostic peculiarities, clinical and radiological diagnostic features, treatment and obstacles to the control of acute leukemia.

The meeting will be open to all physicians concerned with the problem of handling acute leukemia.

TRAINING OF RADIOLOGICAL HEALTH SPECIALISTS

The Department of General Science of Oregon State University in cooperation with the OSU Radiation Center and the Division of Radiological Health of the U. S. Public Health Service has developed a program for the training of Radiological Health Specialists. The curriculum is designed on the basis that integration of related disciplines is the most effective way of developing Radiological Health Specialists. Close coordination among radiation biologists, radiation physicists and public health personnel during seminars and research

conferences is an integral part of the program. A summer course in field radiation surveys will give the student an opportunity to gain experience in the theory and technique of surveys of selected operations, including dental and medical X-ray installations, radioisotope laboratories, nuclear reactors, and other radiation producing devices.

The program may take from 1 to 2 years for completion of the work leading to the M.S. degree in Radiological Health, depending upon the candidate's background.

Deadline for filing applications is April 15, 1964.

For further information and application material write to: Dr. E. Dale Trout, Professor of Radiological Physics, Radiation Center, Oregon State University, Corvallis, Oregon.

SECOND ANNUAL OAK RIDGE RADIOISOTOPE CONFERENCE

Approximately 600 scientists and engineers are expected to attend the Second Annual Oak Ridge Radioisotope Conference to be held April 20–22 in Gatlinburg, Tennessee.

The conference is sponsored by the Oak Ridge Institute of Nuclear Studies (ORINS), Oak Ridge, Tenn., and Oak Ridge National Laboratory, and supported by the United States Atomic Energy Commission.

The program will include more than 40 formal presentations on the uses of radioisotopes in biology, medicine, engineering and in the physical sciences.

Inquiries about the program should be directed to Dr. Ralph T. Overman, Special Training Division, Oak Ridge Institute of Nuclear Studies, P. O. Box 117, Oak Ridge, Tennessee 37831.

POSTGRADUATE COURSE IN PEDIATRIC RADIOLOGY

The Second Postgraduate Course in Pediatric Radiology will be presented at the University of Pittsburgh, School of Medicine, through the sponsorship of the Division of Postgraduate Medicine of the University, April 6, 7 and 8, 1964.

The course will consist of a series of lectures with discussions from the panel participants and from the floor. Emphasis will be placed on "Recent Advances in Pediatric Radiology."

The course will be conducted at the Children's Hospital of the University of Pittsburgh, School of Medicine by an outstanding Faculty of Guest Speakers.

For further information contact: Bertram R. Girdany, M.D., Program Director and Chairman, Postgraduate Course in Pediatric Radiology, Children's Hospital of Pittsburgh, Pittsburgh, Pennsylvania 15213.

It is with deep regret that we announce the death on Friday, March 6, 1964 at Boston, Massachusetts, of Dr. John D. Reeves, Jr., member of the Executive Council of the American Roentgen Ray Society.

BOOK REVIEWS

Books sent for review are acknowledged under: Books Received. This must be regarded as a sufficient return for the courtesy of the sender. Selections will be made for review in the interest of our readers as space permits.

GASTROENTEROLOGY. Volume I. Second Edition. By Henry L. Bockus, M.D., Emeritus Professor of Medicine, University of Pennsylvania School of Medicine; and Present and Former Colleagues at the University of Pennsylvania Graduate School of Medicine. Cloth. Pp. 958, with many illustrations. Price, $25.00. W. B. Saunders Company, West Washington Square, Philadelphia 5, Pa., 1963.

When one attempts to improve on that which is already accepted as a classic in its field, the task is by no means a small one. Volume I of Gastroenterology, which was published by Dr. Henry L. Bockus in 1944, is just such a classic. However, as nothing in this world, and certainly not in the complex field of medicine, is static, it became necessary to modify the old and include the new.

The Second Edition of Volume I has an increased number of pages; 958 as compared to 831 in the first edition. While the number of chapters has remained the same, some have been reorganized, with expansion of their content to include recent advances and deletion of that which is no longer pertinent.

Of particular importance in the Second Edition of Volume I is the fact that many of the chapters or parts of the chapters have been guest-authored by men pre-eminent in their fields. The gastroscopic contribution by Drs. J. F. Monaghan and P. R. Nast is a good example, reflecting the great strides which have been made, since the first printing of the book, in this extremely valuable method of examination including the use of cinegastroscopy and the gastric camera. Perhaps the tremendous effort which has gone into the Second Edition to portray the latest in clinical thought and expanded diagnostic knowledge can best be appreciated by perusing the bibliographies, which have been greatly enlarged and have been laboriously brought up to date.

Areas of particularly significant changes are: the more complete discussion of the physiology and interpretation of pain; the emphasis on a sound clinical approach based on understanding of the gastric physiology and consideration of all possible primary and secondary disorders of the digestive tract and its appendages; and a very comprehensive description of the newer gastric tests.

Space does not permit a detailed enumeration of the many other excellent chapters authoritatively written and profusely illustrated with fine roentgenograms, drawings, photomicrographs and many beautiful figures done in color. The historic, etiologic, pathologic, diagnostic, and therapeutic aspects of all affectations of the esophagus and stomach are lucidly and completely covered.

Dr. Bockus's book is the intellectual gastroscope with which to examine more closely a complex and essential organ system of the human body. It is superbly edited, skillfully composed and artfully printed. This Second Edition of Volume I is unhesitatingly recommended to every physician.

GEORGE A. KLING, M.D.

THE CYTOLOGIC DIAGNOSIS OF CANCER. Second Edition. By Ruth M. Graham, Roswell Park Memorial Institute, Buffalo, New York. Cloth. Pp. 387, with many illustrations. Price, $14.00. W. B. Saunders Company, Philadelphia, Pa., 1963.

The second edition of Dr. Graham's text on the cytologic diagnosis of cancer is welcome and contains some important modifications and additions. Among the latter are new chapters on cell identification, dysplasia of the cervix and cytologic changes in pernicious anemia. The book is intended as an introduction to cytology and has been so used with considerable success by the present generation of pathologists and cytotechnologists. It is easy for a reviewer to find minor faults in any textbook, but there is only one serious defect in this work. The selection of fields for photomicrography of tissue sections leaves much to be desired. Many are taken at too high a power to orient the reader to the spacial arrangement and location of the epithelium. Perhaps future editions might include photographs at more than one magnifi-

cation to cover both pattern and cellular detail. It is especially important for the beginner to correlate the appearance of desquamated cells with the lesion from which they are derived. In this connection more photographs of different patterns seen in dysplasia and carcinoma *in situ* of the cervix would be of value.

In all other respects this book can be highly recommended. The combination of photographs of cells, water color drawings and diagrams presents as lucid an exposition of cell types as is available anywhere. The use of a uniform magnification throughout enhances the value of the photographs by permitting comparison of cell size from page to page.

Dr. Graham writes well. Her style is conversational and forceful. Readers approaching the book for the first time might do well to start with Chapter 18 which deals with the basic principles of cell identification. This chapter is a valuable new addition and might well have been placed at the beginning of the book.

The book is strongly recommended for all physicians or technologists beginning the study of cytology. Other texts are available for those more advanced in the field.

A. R. W. Climie, M.D.

BOOKS RECEIVED

Testo-Atlante di Chimografia Cardiovascolare. By Giovanni Juliani and Cesare Quaglia dell'-Istituto di Radiologia dell'Università di Torino. Cloth. Pp. 218, with 164 illustrations. Price, Lire 10,000. Edizioni Minerva Medica, Torino, Italy, 1963.

Elsevier's Medical Dictionary. In Five Languages, English/American, French, Italian, Spanish and German. Compiled by A. Sliosberg, Consulting Physician of the Institution Nationale des Invalides, Paris, France. Cloth. Pp. 1588. Price, $45.00. American Elsevier Publishing Company, Inc., 52 Vanderbilt Avenue, New York 17, N. Y., 1963.

Symposium on Macromolecular Aspects of the Cell Cycle. Given at Research Conference for Biology and Medicine of the Atomic Energy Commission: sponsored by The Biology Division, Oak Ridge National Laboratory, Oak Ridge, Tennessee. Paper. Pp. 198, with some illustrations. The Wistar Institute of Anatomy and Biology, Philadelphia, Pa., 1963.

Bone Marrow Transplantation after Whole-Body Irradiation: an Experimental Study in the Rat. By Hans Balner, Radiobiological Institute of the Organization for Health Research T.N.O., 151, Lange Kleiweg, Rijswijk Z. H., Netherlands. Paper. Pp. 134, with many illustrations. 1963.

Erweiterungen des Spinalraumes: Radiologische Beurteilung unter besonderer Berücksichtigung der Syringomyelie. By Dozent Dr. med. habil. Peter Hagemann, Oberarzt an der Univ.-Nervenklinik der Charité, Berlin. Paper. Pp. 112, with 35 illustrations. Price, Steif broschiert 16,90 DM. VEB Gustav Fischer Verlag, Jena, Villengang 2/Postschliessfach Mr. 176, Germany, 1963.

Biology of the Prostate and Related Tissues. National Cancer Institute Monograph 12. Edited by Erwin P. Vollmer. Sponsored by Cancer Chemotherapy National Service Center, National Cancer Institute, National Institutes of Health, Bethesda, Md. Cloth. Pp. 446, with many illustrations. Price, $4.00. Superintendent of Documents, U. S. Government Printing Office, Washington, D. C. 20402, 1963.

The Reduction of Patient Dose by Diagnostic Radiologic Instrumentation. Edited by Robert D. Moseley, Jr., M.D., Professor of Radiology, Chairman, Department of Radiology, the University of Chicago, Chicago, Ill., and John H. Rust, D.V.M., Ph.D., Professor of Pharmacology, Head, Section of Nuclear Medicine, the Department of Pharmacology, The University of Chicago, Chicago, Ill. Cloth. Pp. 285, with many illustrations. Price, $12.50. Charles C Thomas, Publisher, 301–327 East Lawrence Avenue, Springfield, Ill., 1963.

The Year Book of Radiology: 1963–1964 Series Radiologic Diagnosis. Edited by John Floyd Holt, M.D., Professor, Department of Radiology, University of Michigan, and Walter M. Whitehouse, M.D., Professor, Department of Radiology, University of Michigan. Radiation Therapy. Edited by Harold W. Jacox, M.D., Professor of Radiology, College of Physicians and Surgeons, Columbia University; Chief, Radiation Therapy Division, Radiologic Service, Presbyterian Hospital, New York, N. Y., and Morton M. Kligerman, M.D., Professor of Radiology and Chairman of the Department of Radiology, Yale University School of Medicine; Radiologist-in-chief, Grace-New Haven Community Hospital. Cloth. Pp. 432, with 332 illustrations. Price, $11.75. Year Book Medical Publishers, Inc., 35 East Wacker Drive, Chicago 1, Ill., 1964.

Clinical Urography: an Atlas and Textbook of Roentgenologic Diagnosis. By John L. Emett, A.B., M.D., M.S. in Urology, Consultant in Urology, Mayo Clinic; Professor of Urology, Mayo Foundation, Graduate School, University of Minnesota, Rochester, Minn. Volume 1. Second edition. Cloth. Pp. 646, with many illustrations. Price, $50.00 the set. W. B. Saunders Company, Philadelphia, Pa., 1964.

SOCIETY PROCEEDINGS

MEETINGS OF RADIOLOGICAL SOCIETIES*

UNITED STATES OF AMERICA

AMERICAN ROENTGEN RAY SOCIETY
Secretary, Dr. C. Allen Good, Mayo Clinic, Rochester, Minn. Annual meeting: Radisson Hotel, Minneapolis, Minn., Sept. 29–Oct. 2, 1964.

AMERICAN RADIUM SOCIETY
Secretary, Dr. Justin J. Stein, U.C.L.A. Medical Center, Los Angeles 24, Calif. Annual meeting: Greenbrier Hotel, White Sulphur Springs, W. Va., April 13–16, 1964.

RADIOLOGICAL SOCIETY OF NORTH AMERICA
Secretary-Treasurer, Dr. Maurice Doyle Frazer, 1744 South Fifty-eighth St., Lincoln, Neb. Annual meeting: Palmer House, Chicago, Ill., Nov. 29–Dec. 4, 1964.

AMERICAN COLLEGE OF RADIOLOGY
Executive Director, William C. Stronach, 20 N. Wacker Drive, Chicago 6, Ill. Annual meeting: Bellvue Stratford Hotel, Philadelphia, Pa., Feb. 10–13, 1965.

SECTION ON RADIOLOGY, AMERICAN MEDICAL ASSOCIATION
Secretary, Dr. Clyde A. Stevenson, Sacred Heart Hospital, West 101 Eighth Ave., Spokane 4, Wash. Annual meeting: San Francisco, Calif., June 21–25, 1964.

AMERICAN BOARD OF RADIOLOGY
Secretary, Dr. H. Dabney Kerr. Correspondence should be directed to Kahler Hotel Building, Rochester, Minn. The Spring 1964 examination will be held at the Americana Hotel, New York City, June 8–12, inclusive. Deadline for filing applications was December 31, 1963. There will be a Special Examination in Nuclear Medicine for diplomates in Radiology or Therapeutic Radiology if sufficient applications are received. The Fall 1964 examination will be held at the Americana Hotel, Bal Harbour, Florida, December 7–11, inclusive. Deadline for filing applications is June 30, 1964.

AMERICAN ASSOCIATION OF PHYSICISTS IN MEDICINE
Secretary-Treasurer, Charles S. Simons, University of Michigan Hospital, Ann Arbor, Mich. Annual meeting to be announced.

AMERICAN CLUB OF THERAPEUTIC RADIOLOGISTS
Secretary, Dr. J. A. del Regato, Penrose Cancer Hospital, Colorado Springs, Colo.

ELEVENTH INTERNATIONAL CONGRESS OF RADIOLOGY
Secretary-General, Professor Dr. Med. Arduino Ratti, via Moscova, 44-1, Milan, Italy. Address inquiries to Professor Dr. Med. Luigi Turano, President-Elect, Istituto di Radiologia, Università di Roma, Rome, Italy. Meeting: September 22–28, 1965.

EIGHTH INTER-AMERICAN CONGRESS OF RADIOLOGY
Counselor for the United States, Dr. Philip J. Hodes, Jefferson Medical College Hospital, 11th and Walnut Streets, Philadelphia 7, Pennsylvania.
Secretary, Dr. Mario Gaitan-Yanguas, Bogotá, Colombia. Meeting to be announced.

ALABAMA RADIOLOGICAL SOCIETY
Secretary, Dr. Walter Brower, Birmingham, Ala. Meets time and place of Alabama State Medical Association.

AMERICAN NUCLEAR SOCIETY
Treasurer, Raymond Maxson, 86 E. Randolph St., Chicago, Ill. Annual meeting to be announced.

ARIZONA RADIOLOGICAL SOCIETY
Secretary-Treasurer, Dr. E. Lawrence Ganter, 1820 E. Polk St., Phoenix 34, Ariz. Two regular meetings a year. Annual meeting at time and place of State Medical Association and interim meeting six months later.

ARKANSAS RADIOLOGICAL SOCIETY
Secretary, Dr. Charles W. Anderson, 1108½ Poplar, Pine Bluff, Ark. Meets every three months and also at time and place of State Medical Association.

ASSOCIATION OF UNIVERSITY RADIOLOGISTS
Secretary-Treasurer, Dr. Sidney W. Nelson. Annual meeting: University of Chicago School of Medicine, Chicago, Illinois, May 14–16, 1964.

ATLANTA RADIOLOGICAL SOCIETY
Secretary, Dr. Donald R. Rooney, Burnt Hickory Road, Marietta, Ga. Meets monthly except during three summer months, on third Tuesday, at the Academy of Medicine, Atlanta, Ga., at 8:00 P.M.

BAVARIAN-AMERICAN RADIOLOGIC SOCIETY
Secretary, Dr. Roy R. Deffebach, Major, MC, Radiology Service, 5th General Hospital, APO 154, New York, N. Y. Meets quarterly.

BLOCKLEY RADIOLOGICAL SOCIETY
Secretary, Dr. Bernard J. Ostrum, 2412 North 52nd St., Philadelphia, Pa.

BROOKLYN RADIOLOGICAL SOCIETY
Secretary, Dr. Peter J. Lampros, 121 Dekalb Ave., Brooklyn, N. Y. Meets first Thursday of each month, October through May.

BUFFALO RADIOLOGICAL SOCIETY
Secretary, Dr. Eugene V. Leslie, Edward J. Beyer Memorial Hosp., Buffalo 15, N. Y. Meets second Monday evening each month, October to May inclusive.

CALIFORNIA RADIOLOGICAL SOCIETY
Secretary, Dr. L. Henry Garland, Suite 1739, 450 Sutter St., San Francisco, Cal. Meets annually during meeting of California Medical Association.

CENTRAL NEW YORK RADIOLOGICAL SOCIETY
Secretary, Dr. Edward W. Carsky, Crouse-Irving Hospital, 820 S. Crouse Ave., Syracuse, N. Y. Meets first Monday each month, October through May.

CENTRAL OHIO RADIOLOGICAL SOCIETY
Secretary-Treasurer, Dr. Harold W. Long, 81 S. Fifth St., Columbus 15, Ohio. Meets third Thursday in October, and second Thursday in November, January, March and May at Fort Hayes Hotel, Columbus, Ohio.

CENTRAL SOCIETY OF NUCLEAR MEDICINE
Secretary, Dr. Robert S. Landauer, Radiation Center Bldg., 1903 West Harrison St., Chicago 12, Ill.

CHICAGO ROENTGEN SOCIETY
Secretary, Dr. Abram H. Cannon, 250 E. Superior St., Chicago 11, Ill. Meets second Thursday of each month, October to April except December at the Pick-Congress Hotel at 8:00 P.M.

CLEVELAND RADIOLOGICAL SOCIETY
Secretary-Treasurer, Dr. James Christie, 10515 Carnegie Avenue, Cleveland, Ohio. Meetings at 7:00 P.M. on fourth Monday of October, November, January, February, March and April.

COLORADO RADIOLOGICAL SOCIETY
Secretary, Dr. David J. Stephenson, 8300 West 38th Ave., Wheat Ridge, Colo. Meets third Friday of each month at Denver Athletic Club from September through May.

CONNECTICUT VALLEY RADIOLOGIC SOCIETY
Secretary, Dr. James L. Krieger, 85 Jefferson St., Hartford 6, Conn. Meets first Friday in February and April.

DALLAS-FORT WORTH RADIOLOGICAL SOCIETY
Secretary, Dr. Paul W. Mathews, Jr., 1217 West Cannon St., Fort Worth, Tex. Meets monthly, third Monday, at Southwest International Airport at 6:00 P.M.

*** Secretaries of societies are requested to send timely information promptly to the Editor.**

Detroit Roentgen Ray and Radium Society
Secretary, Dr. Arch H. Hall, Harper Hospital, Detroit 1, Mich. Meets monthly, first Thursday, October through May, at David Whitney House, 1010 Antietam, at 6:30 P.M.

East Bay Roentgen Society
Secretary, Dr. Dan Tucker, 434 30th St., Oakland 9, Calif. Meets first Thursday each month at Peralta Hospital, Oakland, Calif.

East Tennessee Radiological Society
Secretary, Dr. J. Marsh Frere, Jr., 205 Medical Arts Bldg., Knoxville, Tenn. Meets in January and September.

Eastern Conference of Radiologists
Secretary, Arrangements Committee, Dr. Theodore A. Tristan, 3400 Spruce St., Philadelphia 4, Pa. Annual meeting to be announced.

Eastern Radiological Society
Secretary, Dr. James F. Martin, North Carolina Baptist Hospital, Winston-Salem, N. C.

Florida Radiological Society
Secretary, Dr. David Kirsh, 712 Du Pont Bldg., Miami, Fla. Meets twice annually, in the spring with the annual State Society Meeting and in the fall.

Florida West Coast Radiological Society
Secretary-Treasurer, Dr. Garth R. Drewry, Tampa General Hospital, Tampa 6, Fla. Meets in January, April, July and October.

Georgia Radiological Society
Secretary, Dr. David Robinson, P.O. Box 394, Savannah, Ga. Meets in spring and fall with Annual State Society Meeting.

Greater Miami Radiological Society
Secretary, Dr. Robert F. Feltman, Dade County Medical Association Building, Two S.E. Thirteenth St., Miami 32, Fla. Meets monthly, third Wednesday at 8:00 P.M. at Jackson Memorial Hospital, Miami, Fla.

Greater St. Louis Society of Radiologists
Secretary, Dr. C. M. Witt, #16 Hampton Village Plaza, St. Louis 9, Mo.

Houston Radiological Society
Secretary, Dr. D. A. Van Velzer, Texas Medical Center Library, Jesse H. Jones Library Bldg., Houston 25, Tex. Meets fourth Monday of each month, except June, July, August and December, at the Doctors' Club, 8:00 P.M., Houston, Tex.

Idaho State Radiological Society
Secretary, Dr. George H. Harris, Bannock Memorial Hospital, Pocatello, Idaho. Meets in the spring and fall.

Illinois Radiological Society
Secretary, Dr. George A. Miller, Carle Hospital Clinic, Urbana, Ill. Meets in the spring and fall.

Indiana Roentgen Society, Inc.
Secretary, Dr. Richard A. Silver, 1815 N. Capitol Avenue, Indianapolis, Ind. Meets first Sunday in May and during fall meeting of Indiana State Medical Association.

Iowa Radiological Society
Secretary, Dr. L. L. Maher, 1419 Woodland Ave., Des Moines, Iowa. Luncheon and business meeting during annual session of Iowa State Medical Society. The scientific section is held in the autumn.

Kansas Radiological Society
Secretary, Dr. Roger K. Wallace, Riley County Hospital, Manhattan, Kan. Meets in spring with State Medical Society and in winter on call.

Kentucky Radiological Society
Secretary, Dr. Lawrence A. Davis, 226 East Chestnut St., Louisville, Ky. Meets monthly on second Friday at Sheraton Hotel, Louisville.

Kings County Radiological Society
Secretary, Dr. Stanley Dannenberg, 1917 Bedford Ave., Brooklyn 25, N. Y. Meets Kings County Med. Soc. Bldg. monthly on fourth Thursday, October to May, 8:45 P.M.

Knoxville Radiological Society
Secretary, Dr. Clifford L. Walton, Blount Professional Bldg., Knoxville 20, Tenn. Meetings are held the third Monday of every other month at the University of Tennessee Memorial Research Center and Hospital.

Los Angeles Radiological Society
Secretary, Dr. Bernard J. O'Loughlin, Dept. of Radiology, UCLA Center for Health Sciences, Los Angeles 24, Calif. Meets second Wednesday of month in September, November, January, April and June at Los Angeles County Medical Association Building, Los Angeles.

Maine Radiological Society
Secretary, Dr. Charles W. Capron, Maine Medical Center, Portland, Me. Meets in June, September, December and April.

Maryland Radiological Society
Secretary, Dr. Henry Startzman, Medical Arts Building, Baltimore, Maryland.

Memphis Roentgen Society
Secretary-Treasurer, Dr. Irving K. Ettman, 684 W. Brookhaven Circle, Memphis, Tenn. Meets first Monday of each month at John Gaston Hospital.

Miami Valley Radiological Society
Secretary, Dr. William D. Roberts, 2197 Los Arrow Dr., Dayton 9, Ohio. Meets second Friday of fall and winter months.

Mid-Hudson Radiological Society
Secretary, Dr. Joseph Sorrentino, St. Francis Hospital, Poughkeepsie, N. Y. Meets 8:30 P.M., fourth Wednesday of each month September to May.

Milwaukee Roentgen Ray Society
Secretary-Treasurer, Dr. Harold F. Ibach, 2400 W. Villard Avenue, Milwaukee 9, Wis. Meets monthly on fourth Monday, October through May, at University Club.

Minnesota Radiological Society
Secretary-Treasurer, Dr. Frank J. Anderson, 810 E. 27th St., Minneapolis 7, Minn. Meets three times annually, in fall, winter and spring.

Mississippi Radiological Society
Secretary, Dr. Jack K. Goodrich, University Medical Center, Jackson, Miss. Meets third Thursday of each month at the Heidelberg Hotel, Jackson, at 6:00 P.M.

Montana Radiological Society
Secretary, Dr. Clark Grimm, Great Falls, Montana. Meets at least once a year.

Nassau Radiological Society
Secretary, Dr. Barry L. Lewis, 165 N. Village Ave., Rockville Centre, New York, N. Y. Meets second Tuesday of the month in February, April, June, October and December.

Nebraska State Radiological Society
Secretary, Dr. Richard Bunting, The Radiologic Center, Nebraska Methodist Hospital, Omaha 31, Neb. Meets third Wednesday of each month at 6 P.M. in Omaha or Lincoln.

Nevada Radiological Society
Secretary, Dr. William G. Arbonies, Department of Radiology, St. Mary's Hospital, Reno, Nev.

New England Roentgen Ray Society
Secretary, Dr. Robert E. Wise, 605 Commonwealth Ave., Boston 15, Mass. Meets third Friday of each month, October through April, at The Longwood Towers, 20 Chapel Street, Brookline, Mass., at 4:30 P.M.

New Hampshire Roentgen Ray Society
Secretary, Dr. Paul Y. Hasserjian, 1470 Elm St., Manchester, N. H. Meets four to six times yearly.

New Mexico Association of Radiologists.
Secretary-Treasurer, Dr. Justin J. Wolfson, Department of Radiology, Bernalillo County-Indian Hospital, Albuquerque, New Mexico.

New York Roentgen Society
Secretary, Dr. Francis F. Ruzicka, Jr., St. Vincent's Hospital, New York, 11, N. Y. Meets monthly on third Monday at the New York Academy of Medicine at 4:30 P.M.

Spring Conference: Waldorf-Astoria Hotel, New York, N. Y., April 23–25, 1964.

North Carolina Radiological Society
Secretary, Dr. E. H. Schultz, North Carolina Memorial Hospital, Chapel Hill, N. C. Meets in the spring and fall each year.

North Dakota Radiological Society
Secretary, Dr. John Jestadt, Depuy-Sorkness Clinic, Jamestown, N. D. Meets at time of State Medical Association meeting. Other meetings arranged on call of the President.

North Florida Radiological Society
Secretary, Dr. Charles H. Newell, 800 Miami Road, Jacksonville 7, Fla. Meets quarterly in March, June, September and December.

Northeastern New York Radiological Society
Secretary, Dr. Anthony J. Tabacco, 621 Central Ave., Albany 6, N. Y. Meets in Albany area on second Wednesday of October, November, March and April.

Northern California Radiological Society
Secretary, Dr. Rob H. Kirkpatrick, 1219 28th St., Sacramento, Calif. Meets at dinner last Monday of each month, September to June.

Northwestern Ohio Radiological Society
Secretary. Dr. Vito J. Zupa, Mercy Hospital, Department of Radiology, Toledo, Ohio.

Ohio State Radiological Society
Secretary, Dr. M. M. Thompson, Jr., 316 Michigan St., Toledo 2, Ohio. Annual meeting: Cleveland, Ohio, May 23–24, 1964.

Oklahoma State Radiological Society
Secretary, Dr. Simon Pollack, Utica Square Medical Center, Tulsa, Okla. Meets in January, May and October.

Orange County Radiological Society
Secretary-Treasurer, Dr. Robert M. Sodaro, 600 E. Washington Ave., Santa Ana, Calif. Meets fourth Tuesday of month except June, July, August and December in Orange County Medical Association Building.

Oregon Radiological Society
Secretary-Treasurer, Dr. James A. Schneider, St. Vincent Hospital, Portland 10, Ore. Meets on second Wednesday of month at 7:00 P.M. at the University Club, Portland, Ore.

Orleans Parish Radiological Society
Secretary, Dr. Joseph V. Schlosser, Charity Hospital, New Orleans 13, La. Meets second Tuesday of each month.

Pacific Northwest Radiological Society
Secretary, Dr. Charles E. Donley, 543 East 4th Street, Bend, Ore. Annual meeting on the second weekend in May, 1964.

Pennsylvania Radiological Society
Secretary-Treasurer, Dr. Frederick R. Gilmore, Clearfield Hospital, Clearfield, Pa. Annual meeting: Buckhill Falls, May 21–23, 1964.

Philadelphia Roentgen Ray Society
Secretary, Dr. Antolin Raventos, Hospital of the University of Pennsylvania, Philadelphia 4, Pa. Meets first Thursday of each month at 5 P.M., from October to May in Thompson Hall, College of Physicians.

Pittsburgh Roentgen Society
Secretary, Dr. Robert N. Berk, 230 Lothrop St., Pittsburgh 13, Pa. Meets second Wednesday of month, October through June at Park Schenely Restaurant.

Radiological Society of Connecticut
Secretary-Treasurer, Dr. Orlando F. Gabriele, 1450 Chapel St., New Haven 11, Conn. Meetings are held bimonthly.

Radiological Society of Greater Cincinnati
Secretary, Dr. John F. Popkin, Jr., 831 Carew Tower, Cincinnati, Ohio. Meets first Monday of each month at Cincinnati Academy of Medicine.

Radiological Society of Hawaii
Secretary, Dr. Edgar S. Childs, Kuakini Hospital, Honolulu, Hawaii. Meets third Monday of each month at 7:30 P.M.

Radiological Society of Greater Kansas City
Secretary, Dr. J. Stewart Whitmore, 1010 Rialto Bldg., Kansas City, Mo. Meets last Friday of each month.

Radiological Society of Kansas City
Secretary, Dr. Arthur B. Smith, 800 Argyle Bldg., Kansas City, Mo. Meets third Thursday of each month.

Radiological Society of Louisiana
Secretary, Dr. Andrew F. Giesen, Ochsner Clinic, New Orleans 15, La. Meets annually during Louisiana State Medical Society meeting.

Radiological Society of New Jersey
Secretary, Dr. E. Arthur Kratzman, 912 Prospect Ave., Plainfield, N. J. Meets in Atlantic City at time of State Medical Society meeting and in October or November in Newark, N. J.

Radiological Society of the State of New York
Secretary-Treasurer, Dr. John W. Colgan, 273 Hollywood Ave., Rochester 18, N. Y. Annual meeting to be announced.

Radiological Society of South Dakota
Secretary-Treasurer, Dr. Donald J. Peik, 303 S. Minnesota Ave., Sioux Falls, S. D.

Radiological Society of Southern California
Secretary-Treasurer, Dr. Charles G. Campbell, La Jolla, Calif.

Redwood Empire Radiological Society
Secretary, Dr. Lee F. Titus, 164 W. Napa St., Sonoma, Calif. Meets second Monday every other month.

Richmond County Radiological Society
Secretary, Dr. W. F. Hamilton, Jr., University Hospital, August, Ga. Meets first Thursday of each month at various hospitals.

Rochester Roentgen Ray Society, Rochester, N. Y.
Secretary, Dr. Gerald R. Holzwasser, Rochester General Hospital, Northside Division, 1425 Portland Ave., Rochester 21, N. Y. Meets at 8:15 P.M. on the last Monday of each month, September through May, at Strong Memorial Hospital.

Rocky Mountain Radiological Society
Secretary, Dr. John H. Freed, 4200 East Ninth Ave., Denver 20, Colo. Annual meeting to be announced.

San Antonio-Military Radiological Society
Secretary, Dr. Hugho F. Elmendorf, Jr., 730 Medical Arts Bldg., San Antonio 5, Tex. Meets third Wednesday each month in Fort Sam Houston Officer's Club at 6:30 P.M.

San Diego Radiological Society
President-Secretary, Charles P. Hyslop, 7901 Frost St., San Diego 22, Calif. Meets first Wednesday of each month at the University Club.

San Francisco Radiological Society
Secretary, Dr. Malcolm Jones, University of California Hospital, San Francisco 22, Calif. Meets quarterly at the San Francisco Medical Society, 250 Masonic Ave., San Francisco 18, Calif.

Section on Radiology, California Medical Association
Secretary, Dr. William H. Graham, 630 East Santa Clara St., San Jose, Calif.

Section on Radiology, Medical Society of the District of Columbia
Secretary-Treasurer, Dr. Martin A. Thomas, 1150 Connecticut Ave., Washington 6, D. C. Meets at Medical Society Library, third Wednesday of January, March, May and October at 8:00 P.M.

Section on Radiology, Southern Medical Association
Secretary, Dr. John D. Reeves, Department of Radiology, Univ. of Florida College of Medicine, Gainesville, Fla. Annual meeting to be announced.

Section on Radiology, Texas Medical Association
Secretary, Dr. George F. Crawford, St. Elizabeth Hospital, Beaumont, Tex. Meets annually with the Texas Medical Association.

Shreveport Radiological Club
Secretary, W. R. Harwell, 608 Travis St., Shreveport, La. Meets monthly on third Wednesday at 7:30 P.M., September to May inclusive.

Society for Pediatric Radiology
Secretary, Dr. John L. Gwinn, Children's Hospital, Los Angeles 27, Calif., Annual meeting: Radisson Hotel, Minneapolis, Minn., Sept. 28, 1964.

Society of Nuclear Medicine
Secretary, Mr. C. Craig Harris, Oak Ridge National Laboratories, Oak Ridge, Tenn. *Administrator*, Mr. Samuel N. Turiel, 430 N. Michigan Ave., Chicago 11, Ill. Annual meeting: Claremont Hotel, Berkelev, Calif., June 17–20, 1964.

South Bay Radiological Society
Secretary, Northern Section: Dr. John S. Wilson, 1515 Trousdale Dr., Burlingame, Calif.; Southern Section: Dr. A. C. Mitchell, 728 Cass, Monterey, Calif. Meets second Wednesday of each month.

South Carolina Radiological Society
Secretary, Dr. George W. Brunson, 1406 Gregg St., Columbia, S. C. Annual meeting (primarily business) in conjunction with the South Carolina Medical Association meeting in May. Annual fall scientific meeting at time and place designated by the president.

Southern Radiological Conference
Secretary-Treasurer, Dr. Marshall Eskridge, Mobile Infirmary, P.O. Box 4097, Mobile, Ala. Annual meeting to be announced.

Southwestern Radiological Society
Secretary, John M. McGuire, 904 Chelsea, El Paso, Tex. Meets last Monday of each month at 6:30 P.M. in the Paso del Norte Hotel.

Tennessee Radiological Society
Secretary, Dr. B. M. Brady, St. Joseph Hospital, Memphis, Tenn. Meets annually at the time and place of the Tennessee State Medical Association meeting.

Texas Radiological Society
Secretary, Dr. R. P. O'Bannon, 402 Professional Bldg., 1216 Pennsylvania Ave., Fort Worth 4, Tex. Annual meeting: Houston, Texas, Jan. 29–30, 1965.

Tri-State Radiological Society
Secretary, Dr. John H. Marchand, Jr., Methodist Hospital, Henderson, Ky. Meets third Wednesday of Oct., Jan., March and May, 8:00 P.M., Elks Club in Evansville, Ind.

University of Michigan Department of Roentgenology Staff Meeting
Meets each Monday evening from September to June, at 7:00 P.M. at University Hospital, Ann Arbor, Mich.

Upper Peninsula Radiological Society
Secretary, Dr. A. Gonty, Menominee, Mich. Meets quarterly.

Utah State Radiological Society
Secretary, Dr. Carlisle C. Smith, Salt Lake General Hospital, Salt Lake City, Utah. Meets fourth Wednesday in January, March, May, September and November at Holy Cross Hospital.

Vermont Radiological Society
Secretary, Dr. John R. Williams, 160 Allen St., Rutland, Vt.

Virginia Radiological Society
Secretary, Dr. John M. Ratliff, Mary Immaculate Hospital, Newport News, Va. Interim meeting: Homestead Hotel, Hot Springs, Va., April 24–25, 1964.

Washington State Radiological Society
Secretary, Dr. Robert H. Rosenberg, 7011 38th Ave., N.E., Seattle 15, Wash. Meets quarterly.

West Virginia Radiological Society
Secretary, Dr. Karl J. Myers, The Myers Clinic-Broaddus Hospital, Philippi, W. Va. Meets concurrently with Annual Meeting of West Virginia State Medical Society; other meetings arranged by program committee.

Westchester Radiological Society
Secretary, Dr. Edward T. Bello, Westchester Academy of Medicine, Section on Radiology, Purchase, N. Y. Meets on third Tuesday of January and October and on two other dates.

Wisconsin Radiological Society
Secretary, Dr. Charles Benkendorf, 408 St. Francis St., Green Bay, Wis. Annual meeting each spring in various places.

Cuba, Mexico, Puerto Rico and Central America

Asociación de Radiólogos de Centro America y Panamá. Comprising: Guatemala, El Salvador, Honduras, Nicaragua, Costa Rica and Panamá.
Secretary-General, Dr. Raul Arguello, 5a. Ave., Norte No. 48, San Salvador, El Salvador. Meets annually in a rotating manner in the six countries.

Sociedad de Radiología de El Salvador
Secretary, Dr. Rafael Vaga Gómez.

Sociedad de Radiología de Guatemala
Secretary, Dr. Carlos E. Escobar, 9ª. Calle A 0-05, Zona 1, Guatemala.

Sociedad de Radiología y Fisioterapía Cubana
Secretary, Dr. Miguel A. García Plasencia, Hospital Curie, 29 y F, Vedado, Habana, Cuba. Meets monthly at Curie Hospital.

Sociedad Costarricense de Radiologia
Secretary, Dr. James Fernández Carballo, Apartado VIII, San José, Costa Rica.

Sociedad Mexicana de Radiología, A. C.
Calle del Oro No. 15, México 7, D. F.
Secretary-General, Dr. E. Alvarez Hernández.
Meets first Monday of each month.

Asociación Puertorriqueña de Radiología
Secretary, Dr. R. B. Díaz Bonnet, Suite 504, Professional Bldg., Santurce, Puerto Rico.

Sociedad Radiológica Panameña
Secretary, Dr. L. Arrieta Sánchez, Apartado No. 6323, Panamá, R. de P. Meets monthly in a department of radiology of a local hospital chosen at preceding meeting.

Sociedad Radiológica de Puerto Rico
Secretary, Dr. Jorge Carreras Girard, Suite 504, Professional Bldg., Santurce, Puerto Rico. Meets second Thursday of each month at 8:00 P.M. at the Puerto Rico Medical Association Bldg. in San Juan.

British Commonwealth of Nations

Association of Radiologists of the Province of Quebec
Secretary, Dr. R. Robillard, Notre-Dame Hospital, 1560 Sherbrooke St., East, Montreal, Que., Canada. Meets four times a year.

British Institute of Radiology
Honorary Secretary, Dr. R. D. Hoare, 32 Welbeck St., London, W. 1, England. Meets monthly from October until May.

Canadian Association of Physicists, Division of Medical and Biological Physics.
Honorary Secretary-Treasurer, Paul M. Pfalzner, Dept. of Therapeutic Radiology, University of Western Ontario, London, Ont., Canada. Annual meeting: Halifax, Nova Scotia, June, 1964.

Edmonton and District Radiological Society
Secretary, Dr. J. T. Mason, 540 Tegler Bldg., Edmonton, Alberta, Canada. Meets second Tuesday of each month, October to May.

Faculty of Radiologists
Honorary Secretary, Dr. C. J. Hodson, 47 Lincoln's Inn Fields, London, W.C.2, England. Annual meeting to be announced.

Faculty of Radiologists, Royal College of Surgeons in Ireland
Registrar, Dr. H. O'Flanagan, F.R.C.P.I., D.P.H., 123 St. Stephens Green, Dublin 2, Ireland.

Section of Radiology of the Royal Society of Medicine (Confined to Medical Members)
Meets third Friday each month at 4:45 P.M. at the Royal Society of Medicine, 1 Wimpole St., London, W. 1, England.

Canadian Association of Radiologists
Honorary Secretary-Treasurer, Dr. D. J. Sieniewicz, *Associate Honorary Secretary-Treasurer*, Dr. Maurice Dufresne 1555 Summerhill Ave., Montreal 25, Que., Canada. Annual meeting to be announced.

Montreal Radiological Study Club
Secretary, Dr. Leonard Rosenthall, Montreal General Hospital, Montreal, Que., Canada. Meets first Tuesday evening, October to April.

Section of Radiology, Canadian Medical Association
Secretary, Dr. C. M. Jones, Inglis St., Ext. Halifax, N. S.

Société Canadienne-Française d'Electro-Radiologie Médicale
General Secretary, Dr. Maurice Dufresne, 1560 Sherbrooke (East), Montreal, Que., Canada. Meets third Saturday each month.

Toronto Radiological Society
Secretary, Dr. Wallace M. Roy, St. Joseph's Hospitals, 30 The Queensway, Toronto 3, Ont., Canada. Meets second Monday of each month September through May.

College of Radiologists of Australia
Honorary Secretary, Dr. E. A. Booth, c/o British Medical Agency, 135 Macquarie St., Sydney, N.S.W., Australia.

South America

Asociación Argentina de Radiología
Secretary, Dr. Lidio G. Mosca, Avda. Gral. Paz 151, Córdoba, Argentina. Meetings held monthly.

Ateneo de Radiologia
Secretary, Dr. Victor A. Añaños, Instituto de Radiologia, Santa Fe 3100, Rosario, Argentina. Meets monthly on second and fourth Fridays at 7:00 P.M. in the Hospital Nacional del Centenario, Santa Fe 1300, Rosario.

Colégio Brasileiro de Radiologia
Secretary-General, Dr. Tede Eston de Eston, Caixa Postal 5984, São Paulo, Brazil.

Sociedad Argentina de Radiologia
Secretary, Dr. Armando B. de Onaindia, Santa Fe 1171, Buenos Aires. Meetings are held monthly.

Sociedad Bolivana de Radiología
Secretary, Dr. Javier Prada Méndez, Casilla 1596, La Paz, Bolivia. Meets monthly. General assembly once every two years.

Sociedade Brasileira de Radiologia
Secretary, Dr. Nicola Caminha, Av. Mem. de Sa, Rio de Janeiro, Brazil. General Assembly meets every two years in December.

Sociedade Brasileira de Radioterapia
Secretary, Dr. Oscar Rocha von Pfuhl, Av. Brigadeiro Luiz Antonio, 644, São Paulo, Brazil. Meets monthly on second Wednesday at 9:00 P.M. in São Paulo at Av. Brigadeiro Luiz Antonio, 644.

Sociedad Chilena de Radiología
Secretary, Dr. J. P. Velasco, Avenida Santa María 0410, Santiago, Chile. Meets fourth Friday of each month.

Sociedad Colombiana de Radiologia
Secretary, Dr. Alberto Mejía Diazgranados, Carrera 13, No. 25–31, Apartado aéreo No. 5804, Bogotá, Colombia. Meets last Thursday of each month.

Sociedad Ecuatoriana de Radiología y Fisioterapía
Secretary, Dr. Luis Blum, P.O. Box 3712, Guayaquil, Ecuador.

Sociedad Paraguaya de Radiología
Secretary, Dr. Miguel González Addone, 15 de Agosto 322, Asunción, Paraguay.

Sociedad Peruana de Radiologia
Secretary, Dr. Vicente Ubillus, Apartado 2306, Lima, Peru. Meets monthly except during January, February and March, at Asociación Médica Peruana "Daniel A. Carrión," Villalta 218, Lima.

Sociedad de Radiologia del Atlantico
Secretary, Dr. Raul Fernandez, Calle 40 #41–110, Barranquilla, Colombia. Society meets monthly at the Instituto de Radiologia.

Sociedad de Radiología, Cancerología y Física Médica del Uruguay
Secretary-General, Dr. Ernesto H. Cibils, Av. Agraciada 1464, piso 13, Montevideo, Uruguay.

Sociedade de Radiologia de Pernambuco
Secretary, Dr. Manoel Medeiros, Instituto de Radiologia da Faculdade de Medicina da Universidade do Recife, Caixa Postal 505, Pernambuco, Brazil.

Sociedad de Roentgenologia y Medicina Nuclear de la Provincia de Córdoba
Secretary-General, Dr. Carlos A. Oulton, Santa Rosa 447, Córdoba, Argentina.

Sociedad Venezolana de Radiología
Secretary-General, Dr. Rubén Merenfeld, Apartado No. 9362 Candelaria, Caracas, Venezuela. Meets monthly third Friday at Colegio Médico del Distrito Federal, Caracas.

Continental Europe

Österreichische Röntgen-Gesellschaft
President, Dr. Konrad Weiss, Mariannengasse 10, Vienna 9, Austria. Meets second Tuesday of each month in Allgemeine Poliklinik. Annual meeting: Salzburg, Austria, May 13–15, 1964.

Société Belge de Radiologie
General Secretary, Dr. S. Masy, 256 Chaussée de Wavre, Heverlee-lez-Louvain, Belgium. Meets in February, March, May, June, October, November and December.

Société Française d'Electroradiologie Médicale, and its branches: Société du Sud-Ouest, du Littoral Méditerranéen, du Centre et du Lyonnais, du Nord, de l'Ouest, de l'Est, et d'Alger et d'Afrique du Nord. Central Society meets third Monday of each month, except during July, August and September, rue de Seine 12, Paris, France.
Secretary-General, Dr. Ch. Proux, 9 rue Daru, Paris 8e, France.

Československá Společnost pro Roentgenologii a Radiologii
Secretary, Dr. Robert Poch, Praha 12, Šrobárova 50, Czechoslovakia. Meets monthly except during July, August, and September. Annual general meeting.

Deutsche Röntgengesellschaft
Secretary, Professor Dr. med. H. Lossen, Universitäts-Röntgeninstitut., Lagenbeckstr. 1, Mainz, Germany.

Società Italiana di Radiologia Medica e di Medicina Nucleare
Secretary, Dr. Ettore Conte, Ospedale Mauriziano, Torino, Italy. Meets annually.

Nederlandse Vereniging voor Radiologie
Secretary, Dr. H. F. O. Stricker, Schalklaar, Netherlands.

Scandinavian Roentgen Societies
The Scandinavian roentgen societies have formed a joint association called the Northern Association for Medical Radiology, meeting every second year in the different countries belonging to the Association.

Sociedad Española de Radiología y Electrología Médicas y Medicina Nuclear
Secretary, Dr. D. Aureo Gutierrez Churruca, Esparteros, No. 9, Madrid, Spain. Meets monthly in Madrid.

Schweizerische Gesellschaft für Radiologie und Nuklearmedizin (Société Suisse de Radiologie et de Médecine Nucléaire)
Secretary, Dr. Max Hopf, Effingerstrasse 47, Bern, Switzerland.

Asia

Indian Radiological Association
Secretary, Dr. R. F. Sethna, Navsari Building, Hornby Road, Bombay 1, India.

Indonesian Radiological Society
Secretary, Professor Sjahriar Rasad Taman Tjut Mutiah 1, Diakarta, Indonesia.

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augmented from the literature by instances of primary tumors in recto-sigmoid, tongue, uterus and other organs which metastasize to the bronchus, the necessity for endoscopy with biopsy becomes apparent.

Even tissue studies may not be conclusive, for many tumors, especially the poorly differentiated ones, can histologically resemble primary lung cancer.—*Frank A. Riebel, M.D.*

Fisher, John H. Wegener's granulomatosis: a review of three cases. *Canad. M. A. J.*, Jan. 4, 1964, *90*, 10–14. (From: Department of Pathology, Faculty of Medicine, University of Western Ontario, and The Hamilton King Meek Memorial Laboratory of Pathology, Victoria Hospital, London, Ontario, Canada.)

Wegener's granulomatosis is a fatal disease characterized by necrotizing lesions of the respiratory tract, segmental necrotizing angiitis of blood vessels, and necrotizing glomerulitis which frequently leads to uremia. It is generally believed that the disease is a result of a hypersensitivity antigen-antibody reaction. The respiratory tract, the spleen, the kidneys, and the blood vessels bear the brunt of the disease, with granulomatous ulceration of the respiratory tract being the initial lesion.

Three cases are reported in detail. A roentgenogram of the chest in one patient showed a nodular infiltrate in the right lower lobe. Another patient had multiple rounded opacities in both lungs, having the appearance of multiple abscesses or granulomata. At autopsy, these pulmonary lesions were firm, rubbery, well defined, often with central cavitation.

Correct antemortem diagnosis was made in 2 of the cases. The disease is probably closely related to polyarteritis nodosa.—*Steven H. Cornell, M.D.*

Öztalay, A. Gülen, and Beard, Alice Gamble. Tension pneumothorax: possible etiologic role of high intratracheal pressure. *J. Pediat.*, Oct., 1963, *63*, 530–536. (From: University of Arkansas Medical Center, Little Rock, Ark.)

The authors describe 10 cases of early severe spontaneous tension pneumothorax in the first 6 hours of life, in a series of 2,090 term infants and 365 premature infants. The patient is usually symptomatic in proportion to the amount of free air present. Rapid and massive accumulation of the entrapped air within the first hours of life constitutes a neonatal emergency necessitating familiarity with the physical findings and roentgenologic characteristics in order that therapy may be instituted immediately.

Clinical manifestations became apparent and diagnosis was made between birth and 6 hours of age. Absence of breath sounds and hyperresonance on the affected side seemed pathognomonic for diagnosis of this condition in the delivery room. Early surgical intervention, although in some cases complicated by a mild infiltrative reaction, led to complete recovery of all 6 infants thus treated. Because the majority of these cases had been resuscitated with oxygen from a central source by the use of long No. 12 French catheters for intubation, a study of the role of rate of flow and frequency of interruption of oxygen administration was made. Intratracheal pressures in excess of those necessary for initial expansion were recorded, and it is considered that such high pressures played an important etiologic role in these instances.—*James F. Martin, M.D.*

Neef, W. Beidseitige Pleuraverkalkungen im Alter. (Bilateral pleural calcifications in the aged.) *Fortschr. a. d. Geb. d. Röntgenstrahlen u. d. Nuklearmedizin*, Nov., 1963, *99*, 632–638. (Address: Bezirkskrankenhaus für Tuberkulose und Lungenkrankheiten, Nikolai-Rumjanzew-Strasse 100, Leipzig W 31, Germany.)

Calcific pleural plaques usually are the result of chronic inflammations and of traumatic episodes, like empyema, pneumothorax and hemothorax. They are unilateral and of variable shape. Occasionally, however, bilateral pleural calcifications may also be observed. These can usually be traced to chronic inhalation of asbestos and talcum dust. In rare instances, they can also be observed in persons who had never been exposed to noxious dust. The author, in surveying 111,069 photofluorograms, found 33 such cases with bilateral involvement.

The arrangement of these calcifications usually followed the course of ribs. They were found almost everywhere with the exception of the apical fields. The lungs were free from disease, and there was no evidence of pneumonoconiosis. Only 4 patients were under 65 years of age and two thirds (22) were women.

Etiology and pathogenesis are not clear in view of an entirely negative history. According to the experience of others, the calcifications are confined to the parietal pleura, and the pleural space is not obliterated.

The only outstanding feature is the advanced age which in most of the cases can be made responsible in the absence of other contributing factors. In older people, the pleural surfaces are grayish-white, thickened and not as glittering as in earlier life. Vascular and lymphatic networks of the pleura tend to undergo senile atrophy. This can lead to diminished acid contents of body fluids and to the deposition of calcific material (dystrophic calcifications). The knowledge of such innocuous calcifications is of value in the decision of medicolegal problems.—*E. Kraft, M.D.*

Walzem, David E., and Singleton, Edward B. Tetralogy of Fallot: radiologic evaluation before and after surgical treatment. *Radiology*, Nov., 1963, *81*, 760–769. (From: Departments of Radiology, Texas Children's Hospital and Baylor University College of Medicine, Houston, Texas.)

The authors present a thorough analysis of the pre- and postoperative chest roentgenograms of a group of 67 infants and children with tetralogy of Fallot. Twenty patients were under 1 year of age, 38 were between 1 and 6 years and 9 were 7 and 13 years of age. Of the total group, 22 patients underwent a Blalock-Taussig procedure, 40 a Potts' procedure, and 5 had total corrections. In 13 of the first two operative groups a total correction was performed later as a secondary procedure. Particular attention was directed to the cardiac size and, contour and pulmonary vascularity both before and after operation. The analysis of cardiac size utilized the cardiothoracic ratio with norms selected from the literature.

The preoperative evaluation of this group disclosed that there was no heart enlargement in the patients under 1 year and over 6 years of age. However, 18 of the 38 patients from 1 to 6 years of age had cardiothoracic ratios more than two standard deviations above norms for their age. The reason for this apparent variation between age groups is not clear. The preoperative contour is frequently reported to be normal in infancy, with right ventricular hypertrophy not developing until later in childhood. However, the present study indicates that elevation of the cardiac apex was observed in 92.5 per cent of the total group and that pulmonary vascularity was definitely decreased in 56.7 per cent.

Postoperatively 77.7 per cent of the patients showed an increase in the cardiothoracic ratios within the first 6 months after surgery, while only 22.3 per cent demonstrated either no change or a decrease in this ratio. The increase in the cardiothoracic ratios of the entire series, without regards to age, was 6.9 per cent. The noted increase in heart size in this series correlates with previous reports that an increase in heart size parallels clinical improvement. Nearly all patients demonstrated right ventricular enlargement and 43.3 per cent had an increase in the pulmonary artery segment. Also, 91 per cent showed an increase in pulmonary vascularity.

The case histories of 3 patients, with accompanying roentgenographic reproductions, are presented in detail.—*E. B. Best, M.D.*

Loogen, F. Complications after cardiac surgery. *German Med. Monthly*, Oct., 1963, *8*, 404–412. (Address: Medizinische Klinik der Medizinischen Akademie, Moorenstr. 5, Düsseldorf, Germany.)

The medical aspects of the postoperative cardiac patient have been neglected, probably due to the relatively new field of cardiac surgery and inability to determine long-term results as yet. Arrhythmias and conduction disturbances are fairly common but are not a major problem. Total A–V block is a severe complication but is relatively rare with improved surgical techniques.

Aneurysms due to loosening of sutures occur occasionally after correction of coarctation of the aorta. The patient usually experiences sudden chest pain, collapse and bloody sputum but the aneurysm may be an incidental finding on routine postoperative roentgenograms. Restenosis may occur after correction of a coarctation, thought to be due to progressive thrombosis. It is usually associated with the use of prosthetic materials. Restenosis also occurs after mitral valvular commissurotomy due to reappearance of adhesions or to progression of calcification.

Recanalization of a ductus arteriosus can occur when ligation instead of section has been carried out. It is usually associated with reappearance of symptoms or of objective findings. Pulmonary insufficiency is a rather frequent finding after pulmonary valvotomy. It may appear immediately after surgery or be delayed. The significance of pulmonary insufficiency is not yet determined. The importance of routine postoperative roentgenograms is stressed.—*Barry E. Gerald, M.D.*

Bopp, P., Bussat, P., and Lemonnier, J. Rheumatic heart disease and dextrocardia. *A.M.A. Arch. Int. Med.*, Jan., 1964, *113*, 19–22. (Address: Dr. P. Bopp, Hôpital Cantonal, Centre de Cardiologie, Geneva, Switzerland.)

A 12th case is added to the previously reported cases of rheumatic heart disease in patients with situs inversus. The patient presented had pure mitral stenosis; an unusual aspect of the case was the performance of a trans-septal catheterization which had to be done in a reverse manner from the usual.

The authors do not believe that the course of rheumatic heart disease is modified by the presence of dextrocardia.—*Steven H. Cornell, M.D.*

Varadarajan, M. G. Aneurysm of thoracic aorta with unusual features. *Mediscope*, Madras, June, 1962, *5*, 121–126. (Address: The Government General Hospital, Madras, India.)

A 60 year old male Indian presented with a painful pulsatile mass about his right anterior chest wall which simulated an aneurysm of the ascending aorta. There was no rib erosion evident, however, and there was an unexpected displacement of the trachea and the proximal thoracic portion of the barium filled esophagus to the right, along with narrowing of the

trachea and esophagus which was maximum at the level of the 5th thoracic segment. There was dyspnea on exertion and cough. The vertebrae did not appear involved; the V.D.R.L. was strongly positive.

Postmortem examination revealed the right anterior chest wall swelling to be made up of blood clot covered by bare skin which was due to a small leak from the aorta. The tracheal and esophageal findings were due to an uncommonly situated luetic saccular aneurysm which was located in the posterior wall of the ascending aorta just above the aortic cusps and extended towards the vertebral column. There was also a sacculo-fusiform aneurysmal dilatation of the arch of the aorta particularly in its posterior portion.

It is pointed out that the more common type of saccular aneurysm of the ascending aorta is one of "physical signs" as it enlarges towards the right anterior chest wall, and if large enough will erode the second and third ribs and press out through the sternoclavicular joint. Aneurysms of the aortic arch are usually aneurysms with "symptoms" due to pressure on one of the intimately adjacent structures such as the trachea, esophagus, thoracic duct, left main stem bronchus, left recurrent laryngeal nerve, or vertebral bodies. It is also noted that combined aneurysmal involvment of the ascending and transverse portions is not uncommon.—*Donald M. Monson, M.D.*

Delvigne, J. L'hémangiome mediastinal. (Mediastinal hemangioma.) *J. belge de radiol.*, 1963, *46*, 452–461. (Address: Service de Radiodiagnostic, Hôpital de Bavière, Université de Liège, Liège, Belgium.)

Benign mediastinal tumors are comparatively rare, and of these only 2 to 3 per cent are lymphangiomas or hemangiomas.

The author's case of cavernous hemangioma was located in the posterosuperior mediastinum. It was slow-growing, did not pulsate, and was differentiated from aortic or pulmonary artery aneurysm by angiography; there was no souffle or thrill. The large mass was excised together with part of the aorta, and an aortic graft successfully placed. Source of blood supply of the tumor was via the intercostal arteries. —*Frank A. Riebel, M.D.*

Berman, Barnett. Pulmonary hypertrophic osteoarthropathy. *A.M.A. Arch. Int. Med.*, Dec., 1963, *112*, 947–953. (Address: 714 Park Avenue, Baltimore 1, Md.)

The syndrome of hypertrophic pulmonary osteoarthropathy consists of: 1. periosteal proliferation of the distal ends of the long bones; 2. joint manifestations of swelling, pain and tenderness; and 3. clubbing of the fingers.

Chronic pulmonary disease has been the most frequent cause of osteoarthropathy. In the early part of this century chronic lung abscess, empyema and infected bronchiectasis were the most frequent associated findings. The presence of pulmonary tuberculosis without complicating bronchiectasis, lung abscess or cyanosis is almost never associated with this syndrome. It is also seen in congenital heart disease with right to left shunt and cyanosis, in pulmonary arteriovenous fistulae, in subacute bacterial endocarditis, and, less frequently, in chronic lung disease, steatorrhea, myxedema, chronic myeloid leukemia, carcinoma of the nasopharynx and carcinoma of the thymus. The greatest relative frequency of association of hypertrophic osteoarthropathy is with a mesothelioma involving the visceral pleura, where a 50 per cent incidence is reported. It occurs in less than 5 per cent of cases with bronchogenic carcinoma.

A case is presented in which the arthropathy preceded the appearance of a bronchogenic carcinoma by 2 years.

Experimental and clinical evidence indicates that an increased peripheral blood flow is undoubtedly present but there must be some other unknown neurogenic or endocrine causative factor.—*David Morse, M.D.*

Genitourinary System

Haage, H., and Rehm, A. Spasmus der Nierenarterien bei der Renovasographie. (Spasm of renal arteries during renal vasography.) *Fortschr. a. d. Geb. d. Röntgenstrahlen u. d. Nuklearmedizin*, Nov., 1963, *99*, 649–653. (Address: O. A. Dr. H. Haage, Röntgenabteilung, Knappschaftskrankenhaus, 4600 Dortmund, Germany.)

Spasm of a renal artery is a rare condition so that its existence is doubted. The authors, however, report two cases in which they could observe this phenomenon.

In the first case, aortography revealed a small segment of irregular narrowing of the renal artery distal to the branching of the suprarenal artery. Slight arteriosclerotic changes were also observed in this area. Subsequent selective arteriography with the red Ödman catheter corroborated the previously suspected stenosis and disclosed an additional ring-like incisura more distally. A third injection of contrast material after withdrawal of the catheter tip now revealed a normal width of the renal artery.

In the second case, aortography disclosed poor vascularization of the dorsal branch of the left renal artery with diminished opacification of the lower pole of the left kidney. Selective arteriography demonstrated irregular filling of the distal portion of the dorsal branch. On repeated examination there was complete closure of this branch without irritation of other vessels. A nephrectomy was performed,

which revealed a marked infectious process of the lower pole of the left kidney with perirenal adhesions and cortical necrosis in spite of patent arteries.

In the first case, mechanical irritation of the catheter tip contributed to the vasospasm. Arteriosclerotic changes were also present in the more proximal parts of the artery. The pressure of the injection could have been another contributing factor, likewise a chemical irritation of the injected iodine solution.

In the second case, there was absence of an organic obstruction, of arteriosclerotic changes, and of thrombangiitis. Therefore, an isolated angiospasm would have to be assumed as an explanation of the anemic infarct and secondary cortical stenosis.

It is concluded that the contrast material could cause a mechanical and chemical irritation. In addition, vasospasm could be induced by the drug and by a damaged vessel. Most likely, a combination of at least two of these factors is responsible. Serial angiograms are necessary to prove the transient nature of the spasm.—*E. Kraft, M.D.*

Kong, Thomas Quin, Meaney, Thomas F., Dustan, Harriet P., and Sones, F. Mason. Safety of selective renal arteriography. *Am. J. M. Sc.*, Nov., 1963, *246*, 527–531. (From: Research Division, the Division of Radiology and Department of Pediatrics and the Cardiac Laboratory, Cleveland Clinic Foundation, Cleveland, Ohio.)

In selective renal arteriography contrast material is injected directly into a renal artery. To determine the safety of this technique urinary protein excretion was measured before and after arteriography in 31 patients. Because Kirkland and Haslock have reported that proteinuria can follow the intravenous or intraarterial injection of contrast material, the results of 31 patients were compared with those obtained in 17 patients before and after left ventriculography and ascending aortography.

Following the introduction of the catheter into the femoral artery by the Seldinger technique, selective renal arteriography was performed by injecting 4 to 6 ml. of 50 per cent hypaque sodium by manual injection. Additional injections were made if needed. At least one aortic injection was performed to visualize the orifices of the renal arteries and to determine their total number; 10 to 20 ml. of contrast material was injected with an autopressure injector, at a pressure of 75 lb. per square inch. Care was taken to direct the catheter tip away from the renal arteries.

The volume of 90 per cent hypaque sodium used during cinecardioangiography ranged from 76 to 196 ml. and averaged 120 ml.

Renal arteriography produced no changes in renal function except in 1 patient with essential hypertension who had nonocclusive bilateral renal arterial lesions. Prior to arteriography urinary protein excretion had been normal but immediately following the test it was found to be 1.04 grams for 24 hours. Subsequent measures gave normal values. In 6 patients who had abnormal excretion rates before arteriography, proteinuria was not increased afterward. Urea and creatinine clearance were also unaffected.

Cinecardioangiography did not affect the normal protein excretion in 16 patients. In 1 with elevation of the urinary protein before the test an increase was noted after the study. This returned to the pre-test range 1 month later.—*David Morse, M.D.*

Sutton, David, Brunton, F. J., Foot, E. C., and Guthrie, J. Fibromuscular, fibrous and non-atheromatous renal artery stenosis and hypertension. *Clin. Radiol.*, Oct., 1963, *14*, 381–391. (From: Departments of Radiology, and Pathology, St. Mary's Hospital, London, W.2, England.)

In recent years "fibromuscular hyperplasia" of the renal arteries has been established as a pathologic entity causing renal artery stenosis and secondary hypertension. With the increasing use of arteriography in hypertension many more cases due to atheromatous and nonatheromatous lesions are being discovered. Among some 500 hypertensive patients there have been over 60 cases of atheromatous stenosis. Thirty-five cases of atheromatous stenosis have been encountered in patients with aortic or peripheral vascular disease. Thirty-five cases of non-atheromatous renal artery stenosis have been seen and the purpose of this paper is to discuss the non-atheromatous group of cases in detail.

In fibromuscular hyperplasia clinically there is evidence of renal ischemia with secondary hypertension, due to renal artery stenosis. The disease is predominantly female in sex incidence, the majority of the authors' patients being over 40 years of age. Definitive roentgen diagnosis depends upon arteriography. The pyelographic syndrome of renal artery stenosis in its extreme form consists of reduction in size of the affected kidney, increased contrast density in the affected kidney with "spasticity" of the calyces in this kidney, smooth contour of the small kidney in contrast to the small irregular contracted kidney of chronic pyelonephritis, and delayed excretion effect on the affected side. Fibromuscular hyperplasia is often bilateral, being unilateral in only 14 of the authors' 26 cases. Fibromuscular hyperplasia must be producing true renal artery stenosis and ischemia before hypertension will ensue. It tends to involve the middle and distal thirds of the main artery, while atheromatous stenosis involves the origin or proximal centimeter of the renal artery. Two cases of fibrous stenosis of the renal artery are reported in this paper together with 2 cases of extrinsic stenosis from fibrous bands. Two cases of congenital renal

artery hypoplasia: 1 in infancy and 1 at the age of 2 years, are also reported. Pathology of the constricting lesion was available in 7 of the 35 cases of nonatheromatous renal artery stenosis. The final histologic diagnosis was medial fibromuscular hyperplasia (2 cases), perimuscular fibrosis (2 cases), localized congenital hypoplasia (1 case) and extrinsic pressure bands (2 cases). In the extrinsic pressure cases histologic study revealed that the bands contained sympathetic fibers and appeared to be associated with the lumbar sympathetic chain.—*Samuel G. Henderson, M.D.*

KRETCHMAR, L. H., GREENE, W. M., and MCDONALD, D. F. Evaluation of renal function in shock with the radioisotope renogram. *Surg., Gynec. & Obst.*, Dec., 1963, *117*, 733–737. (From: Buswell Urology Research Laboratory, University of Rochester School of Medicine and Dentistry, Rochester, N. Y.)

Using heparinized female dogs, renograms, urine flow rates and urine osmolalities were obtained before bleeding, after blood loss and during severe shock with the blood pressure 50 mm. of mercury or less. During this latter stage, several drugs were given and the effect on the urine flow and the renogram noted. The isotope used was I^{131} hippuran in a dose of 1 $\mu c/3$ kg.

The normal dog renogram is similar to that of the human. The renogram obtained in hypovolemia was that of a higher, broader curve, comparable to tracings obtained in dehydrated animals. The renogram obtained with the animal in shock was similar to that seen with an obstructed kidney, showing a slow ascending curve, due to an accumulation of I^{131} hippuran in the tubules. A sharp descent in the curve was noted as the radioactive material leaves the kidney after injection of a diuretic agent.

The drugs used were levarterenol, metaraminol, mannitol, and THAM. The last was the most effective drug for inducing diuresis during hypovolemic shock. This drug is an osmotic diuretic as well as a potent intracellular buffering agent. Its effect on renal function and shock is related to these factors and perhaps others as yet unknown.—*Barry E. Gerald, M.D.*

BROWN, FRANK A., GELBER, ROBERT H., YOUKELES, LEE H., and BENNETT, LESLIE R. Quantitative approach to the I^{131} renogram. *J.A.M.A.*, Oct. 19, 1963, *186*, 211–214. (From: Departments of Radiology and Preventive Medicine and Public Health, University of California School of Medicine, Los Angles, Calif.)

The quantitative parameters $T_{max.}$ and $T_{\frac{1}{2}}$, previously described by Stewart and Haynie, as well as $T_{max.}$ and/or $T_{\frac{1}{2}}$ and $T_{max.}$ and $T_{\frac{1}{2}}$ were utilized in the interpretation of the radioisotopic renogram as an aid in distinguishing normal from abnormal kidneys.

$T_{max.}$ is defined as the time from point of injection to point of maximum count rate. $T_{\frac{1}{2}}$ is the time from point of maximum count rate to the point where the count rate has fallen to one-half its maximum value.

Values of less than 4 min. for the $T_{max.}$ and less than 7 min. for the $T_{\frac{1}{2}}$ were chosen after renograms on 22 normal subjects were studied. With these values false positives were reduced to 5 per cent for the $T_{max.}$ and 16 per cent for the $T_{\frac{1}{2}}$.

Patients with renal artery lesions, glomerulonephritis, pyelonephritis and obstructive uropathy were studied. Although the number of cases in each category is relatively small it is felt that this approach can detect major kidney disease with reasonable accuracy except for acute and subacute glomerulonephritis.—*Philip Rubin, M.D.*

STOKES, JAMES M., and TER-POGOSSIAN, MICHEL M. Double isotope technique to measure renal functions: single injection technique without urine collection. *J.A.M.A.* Jan. 4, 1964, *187*, 20–23. (From: Department of Surgery, Washington University School of Medicine, and the Mallinckrodt Institute of Radiology, St. Louis, Mo.)

Simultaneous double tracer studies represent an interesting application of radioisotope techniques. Perhaps the best known member of this family of tests is the simultaneous estimation of plasma and blood cell volume. In the present study, however, two isotopic forms of the same element, iodine, namely I^{125} and I^{131}, have been used to estimate the distribution of very similar chemical substances in the blood.

The techniques of gamma spectrometry are readily employed with these two isotopes because of the considerable difference in the qualities of their gamma-ray emissions. I^{125} principally emits a 27.4 kev. K x-ray, and a gamma ray at 35.4 kev. The major I^{131} peaks at 364 kev. and 638 kev. are well known. The gamma-ray spectrometer used to count these two emitters simultaneously provides a window at from 20 to 60 kev. and a second window from 80 kev. to infinity. A sample of I^{125} counted in such a spectrometer with the first window open will be counted in the first window but will show no activity in the second, or higher, window. I^{131}, however, will be counted at full efficiency in the second window, but will also show some count in the first window representing scatter and degraded radiations.

If the counting rate in the lower, or I^{125} window, is A and the counting rate in the higher, or I^{131} window, is B then the amount of I^{125} in the sample is proportional to:

$$A - B \times \frac{(A)}{(B)} I^{131}.$$

In this particular study the authors have employed the dual tracer method to study simultaneously the distribution of sodium diatrizoate tagged with I^{131} and sodium iodohippurate tagged with I^{125} and have estimated their excretion kinetics on the basis of whole blood activities found in samples obtained in from 5 to 60 minutes after injection of the tracer materials. The technique will lend itself to the study of similar complex physiologic functions.—*Frederick J. Bonte, M.D.*

Nervous System

Lodge, Thomas. Radiology in the management of paraplegia. *Clin. Radiol.*, Oct., 1963, *14*, 365–380. (From: Lodge Moor Hospital, Sheffield, England.)

The large majority of Lodge's 320 cases surveyed for this study were men (rarely women), in the prime of life with paraplegia of sudden onset due to spinal injury. With modern control of inevitable infections and a better understanding of nursing requirements, such patients can now be saved from early death and taught to help themselves and to re-educate their locomotor and excretory systems. The mortality of "broken neck" patients has now been reduced from 45 to 8.4 per cent. Almost all cases are due to direct violence, usually hyperflexion. The type of resultant fracture depends on whether or not the posterior ligament complex is ruptured. The commonest level of fracture is between the 11th thoracic and the 2nd lumbar vertebrae. If rupture of the posterior ligament has occurred, the adjacent spinous processes will be seen to be abnormally separated vertically on roentgenograms made in the lateral projection.

Four structures are involved in severe spinal injuries: the vertebral body; the intervertebral disk; the intervertebral (apophyseal) joints; and the interspinous ligament. As the injury heals the vertebral body increases in density, the fracture is obliterated and anterior and lateral ossification takes place.

Cervical injuries. Puzzling fractures may occur in the cervical spine, often associated with tetraplegia. Extension injuries and so-called "whiplash" injuries may show no definite fracture. Flexion fracture-dislocations are usually seen at the 5th cervical vertebra. Vertical sagittal fractures of a single vertebral body tend to occur in the 4th or 5th vertebra and are invariably confused with the air-filled slit of the larynx. These fractures almost always result in paraplegia. Rheumatoid disease may predispose to hematomata in and around the spinal cord and in some cases fracture is found after injury, but the cervical cord may be irretrievably damaged, and the patient die.

Management. During the healing stage of the spinal injury many services have to be carried out by the nursing staff. There is a definite association between bowel stasis and urinary infection. In general these patients show no delay in passage of a barium meal until it reaches the region of the splenic flexure of the colon.

The urinary system. Avoidance or immediate treatment of infection and adequate emptying of the bladder are extremely important in prevention of back pressure dilatation of the upper urinary tracts, with the risk of pyelonephritis. Catheter drainage, unless properly performed, may lead to fistulae, sinuses and/or urethral diverticula in male paraplegics, subsequent to periurethral abscess. Vesicoureteric reflux is an important but late complication. It is commoner in higher infections but is reversible if efficient bladder emptying can be established.

The locomotor system. The most striking roentgenologic manifestations in paraplegic patients are to be seen in the bony skeleton and the soft parts below the level of the spinal lesion. The commonest occurrence is osteoporosis, which is almost invariably seen in the leg bones, and usually also in the pelvis. When osteoporosis in the leg bones is present it is most marked around the knee joint. It can be extreme in the foot. Erosion of bones takes place over the pressure points. When new bone is formed over the ischia it has a "whiskered" appearance.

Changes in joints. There is often gross calcification around a joint but usually little or no change within the joint. In the sacroiliac joints, frequently, there may be seen changes consisting of narrowing of the joint spaces with small articular erosions, similar to the appearance seen in ankylosing spondylitis.

Changes in soft tissue. Ossification and calcification in the muscular tissues below the level of the spinal lesion may be noted. These changes are hardly ever above the pelvis or below the knee. Ossification around the hips may be so massive as to obstruct flexion and may have to be removed. Ossification around the knees may resemble ectopic bone known as the Pellegrini-Stieda lesion. As with bone changes, so with soft tissue ossification; it is rarely seen in the upper extremity, even with cervical lesions. No satisfactory explanation for this ossification in soft tissues has been proposed. Recumbency promotes the mobilization of calcium from the bones, and this may be an etiologic factor. It is less common in recent years, possibly as a result of modern methods of treatment.—*Samuel G. Henderson, M.D.*

Varadarajan, M. G. Varied neurological features in skeletal fluorosis. *Mediscope*, Madras, Aug., 1962, *5*, 335–342. (Address: The Government General Hospital, Madras, India.)

Neurologic abnormalities in skeletal fluorosis are attributed to osseous proliferation about the skull and the vertebral column with resultant pressure on the nerve roots and also the spinal cord from a decrease in the diameter of the spinal canal by bony ingrowths into the canal. Reported neurologic deficits include generalized muscular wasting, muscular

tenderness on pressure, complete rigidity of the spine, brisk knee and ankle jerks, bilateral plantar response, loss of vibratory sense in the lower extremities, and patchy anesthesia. There may be features of paroxysmal headaches, meralgia paraesthetica, Brown-Sequard syndrome, root compression, and amyotrophic lateral sclerosis. The chief neurologic lesion is the development of a spastic paraplegia in extension which slowly changes to a paraplegia in flexion. A spinal sensory level is present in most of the cases. After a fairly prolonged intoxication due to excessive amounts of fluorine, there is considerable invalidism so that the helpless victims of this disease cannot even stand without support. Ultimately they develop crippling deformities and are completely bed-ridden.

The included case report is that of a 45 year old Indian woman presenting with a picture of spastic paraplegia, with an L1 sensory level and perianal anesthesia due to spinal compression.

It is pointed out that it takes anywhere from 1 to 40 years of residence in an endemic area for a definite picture of skeletal fluorosis to develop, depending on how high the fluorine content of the water, how excessively warm the climate, and how poor the state of nutrition. From childhood to about 30 years of age only mottling of the teeth is noted, with few other ill-effects exhibited. It is after this that symptoms of intoxication appear. Diets which include one or more deficiencies of animal proteins, fats, calcium, phosphorus, and vitamins A, C, and D, in the presence of a high fluorine content of water appear to favor the development of crippling deformities and disabilities in skeletal fluorosis. The degree of disability is related to the amount of physical strain, with pain and stiffness being more severe in the joints used most by the individual.

The author also mentions again that the low concentration of one part of fluorine per million parts of water, which is effective in lowering the incidence of dental caries, is innocuous.—*Donald M. Monson, M.D.*

Skeletal System

Sacrez, Ch. Gros. R., Lévy, J. M., Walter, J. P., and Francfort, F. F. (Strasbourg, France.) Hypertélorisme avec malformations cranio-faciales encéphaliques et vertébrales multiples. (Hypertelorism with multiple cranio-facial encephalic and vertebral malformations.) *J. de radiol., d'électrol. et de méd. nucléaire,* Oct., 1963, 44, 635–638.

The authors report the case of a 19 month old boy with hypertelorism without craniostenosis. There was markedly retarded psychomotor development and multiple developmental anomalies and malformations. These included trochocephaly with marked brachycephaly, posterior midline cyst with partial agenesis of the corpus callosum as demonstrated by pneumoencephalography, deformity of the nose and of the mouth, malposition of the teeth, harelip, multiple anomalies of the posterior arches of the dorsal vertebrae, flattening of the costovertebral angle with the ribs almost horizontal, left cryptorchidism, marked laxity of the joints, muscular atrophy and definite retardation of bone maturation.

Of considerable interest in this case was the genetic history. The maternal grandmother, the mother, a brother and a half sister had the same condition with associated mental retardation.

Evidently this is a dominant condition transmitted on the maternal side.

Six photographs and reproductions of roentgenograms accompany this case report.—*William H. Shehadi, M.D.*

Alonso, Francisco Arce, and Ochoa, Manuel Arce. Importancia del diagnóstico radiológico en la enfermedad de Albers-Schoenberg. (Importance of diagnostic radiology in Albers-Schönberg's disease.) *Acta ibér. radiol. cancerol.*, July–Sept., 1963, *18*, 259–272. (From: Hospital del Niño Jesús y Hospital Asilo de San Rafael, Madrid, Spain.)

Osteopetrosis is a familial disorder usually manifested in the first 4 years of life (sometimes *in utero*) and is characterized clinically by anemia, hepatosplenomegaly, and occasionally lymphadenopthy. Roentgenologic examination of the skeleton is necessary for diagnosis and for following the individual patients.

There is sclerotic change of the bones involving mainly the metaphyses and diaphyses. The base of the skull is sclerotic and there may be impingement upon the foramina. Occasionally the facial bones are involved. The long bones, spine, pelvis and ribs are almost uniformly involved. The degree of involvement may vary from punctate areas of sclerosis around the metaphyses to transverse bands of increased opacity, linear areas of sclerosis in the long bones and ribs, or a rather general increase in radiopacity of these bones. The vertebral bodies may present a "sandwich" appearance with relatively greater sclerosis of the superior and inferior borders. The bones of the pelvis and sometimes the ends of the long bones may present the appearance of "a bone within a bone." The metaphysis of long bones may be somewhat expanded and demonstrate uniform sclerosis or alternating transverse bands of sclerosis and areas of lesser opacity. In the posteroanterior view of the skull the sclerosis around the orbits may resemble the appearance of goggles.

Two cases are reported with demonstration of roentgenologic changes throughout the skeleton.—*Charles M. Nice, Jr., M.D., Ph.D.*

Rosensweig, J., Pintar, K., Mikail, M., and Mayman, A. Benign osteoblastoma (giant osteoid osteoma): report of an unusual rib tumour and review of the literature. *Canad. M.A.J.*, Dec. 7, 1963, *89*, 1189–1192. (From: Departments of Cardio-Thoracic Surgery, Pathology and Medicine, Jewish General Hospital, Montreal, Quebec, Canada.)

The authors detail the findings in a patient with a large sclerotic osteoblastoma in a rib. The lesion was found on a routine roentgenographic chest examination in a male aged 27. He reported only occasional slight chest pain on the right. The tumor expanded the posterior portion of the sixth right rib and was densely sclerotic. The adjacent ribs were notched. Chest and bone survey studies were negative.

This is an unusual roentgen appearance, as these lesions commonly are osteolytic, with thinning or erosion of the overlying cortex and varying degrees of sclerosis. Symptoms are few even though the lesions may be large.

Surgery in this case disclosed a stony-hard mass without soft tissue involvement and a wide excision was done because of the clinical and roentgen suspicion of osteogenic sarcoma of the parosteal type. Pathologic examination showed no infiltration of the soft-tissue, but the adjacent muscles were compressed and the parietal pleura thickened.

These lesions are histologically related to osteoma and osteoid osteoma with osteoblastic proliferation and osteoid formation in a vascular stroma, although the roentgen findings are dissimilar. The term osteoblastoma was introduced by Jaffe in 1956. The authors list 43 reported cases and this is the third such reported case in a rib.

Osteoid osteoma and osteoblastoma have been classified as tumors, but the authors suggest that locally altered blood supply (cause?) may stimulate osteoblastic activity and result in these lesions.—*Martha Mottram, M.D.*

Kallio, Erkki. Osteoid osteoma of the metacarpal and metatarsal bones. *Acta orthop. scandinav.*, 1963, *33*, 246–252. (From: Clinic for Orthopaedics and Traumatology, University of Helsinki, Helsinki, Finland.)

Although osteoid osteoma has been reported in almost every bone in the skeleton, the metacarpals and metatarsals are unusual sites for these lesions. In 1959, Freiberger collected 425 cases from the literature and added 80 of his own, and of these 505 cases only 3 were in the metacarpals and 3 in the metatarsals.

The present report deals with:

1. A 20 year old female with swelling of the dorsum of the hand for 2 years and persistent pain and tenderness over the distal end of the third metacarpal. The shaft showed fusiform expansion, thickening and marked sclerosis with a small nidus in the third metacarpal.

2. A 20 year old male with pain, swelling and tenderness for 1 year over the dorsum of the foot. The shaft of the second metatarsal showed periosteal new bone formation and the thickened cortex obscured the nidus.

Both cases had surgery with histologic verification. Interestingly, the first case was clinically diagnosed and treated for bone tuberculosis for 8 months and the second was considered a march fracture initially.—*Martha Mottram, M.D.*

Barnes, William C., and Malament, Maxwell. Osteitis pubis. *Surg., Gynec. & Obst.*, Sept., 1963, *117*, 277–284. (From: Urology Section, Surgical Service, Veterans Administration Hospital, East Orange, N. J.)

Osteitis pubis is a disease affecting the pubic bone and its rami. It usually follows open surgery to the prostate or bladder regions. Rarely it has been seen without previous surgery.

Characteristically the onset is between 2 weeks to 3 months following surgery. It begins with acute pain over the symphysis pubis and the inner aspects of the thighs. There is point tenderness over the symphysis. The pain increases and walking becomes very painful. Flexing the thighs helps relieve the pain. There may be an accompanying low grade fever.

Roentgenograms may not show any abnormality until 2 to 4 weeks after the onset of symptoms; then a moth-eaten rarefaction of the symphysis, loss of trabeculation and eventually separation of the symphysis will develop.

Differential diagnosis includes osteomyelitis and metastatic tumor. Osteomyelitis produces the same symptoms but is usually associated with high and prolonged pyrexia. Metastatic disease may be excluded on a clinical basis.

Various treatments have been used. The authors obtained very good results in 5 cases with nonhormonal, antiinflammatory drugs—phenylbutazone and oxyphenbutazone. They believe that a combination of trauma, infection and venous stasis tends to produce this disease.—*George L. Sackett, Jr., M.D.*

Salvati, Augustín A. (Buenos Aires, Argentina). Artrografia en la displasia luxante de la cadera. (Arthrography in luxative dysplasia of the hip.) *Rev. ortop. y traumatol.* (Latino Americana), July, 1963, *8*, 101–112.

A series of 776 patients with congenital luxation of the hip was observed over a period of 27 years. In the first 14 years there were 400 patients seen before the use of arthrography. During the last 13 years 376 have been studied by means of arthrography to aid in the evaluation and treatment of this condition.

At first water soluble contrast agents in approximately 25 per cent concentration were used. Usually

3–4 ml. is sufficient for this examination. During the past year air has been used in quantities of 5–10 ml. and at present this is the contrast medium of choice. A thin needle is introduced anteriorly or from the anterial inferior direction. A solution of sterile saline in anesthetized infants or of 1 per cent novocain in older patients under local anesthesia may be used to test whether the needle is within the joint space.

Arthrography helps to bring out detail concerning the femoral head, the articular cartilage and the joint capsule. It has also proved very valuable in demonstrating when the reduction is adequate. The inferior capsule has been found to interfere with reduction in the majority of cases presenting difficulty with reduction. An isthmus may be formed between the superior and inferior margins of the capsule. A narrow isthmus may interfere completely with reduction. The teres ligament and interposition of fibrocartilage also interfere with reduction. Osseous deformity and occasionally hypertrophy of the transverse ligament of the acetabulum or excess of fibroadipose tissue in the acetabular fossa may similarly create difficulties.

Arthrography should be considered as a routine procedures in patients between 1 and 3 years of age. It is not generally used in children under 1 year of age, with the exception of some unusual embryonic or teratologic cases. After 3 years of age the possibility of reduction can be evaluated clearly by the usual clinical roentgenologic examination.—*Charles M. Nice, Jr., M.D., Ph.D.*

Bland, John H., Davis, Philip H., London, Marshall G., Van Buskirk, Frederick W. and Duarte, Cristobal G. Rheumatoid arthritis of cervical spine. *A.M.A. Arch. Int. Med.*, Dec., 1963, *112*, 892–898. (From: Rheumatism Research Unit, Departments of Medicine, Surgery, and Radiology, University of Vermont College of Medicine, Burlington, Vt.)

Eighty-six of one hundred cases of definite rheumatoid arthritis had roentgenologic evidence of cervical spine involvement.

Ten roentgenologic criteria for rheumatoid arthritis of the cervical spine are listed: 1. atlanto-axial subluxation of 2.5 mm. or more; 2. multiple subluxations of C_{2-3}, C_{3-4}, C_{4-5} and C_{5-6}; 3. narrow disk spaces with little or no osteophytosis, which are considered (a) pathognomonic at C_{2-3} and C_{3-4}, and (b) probable at C_{4-5} and C_{5-6}; 4. erosions of vertebrae, especially vertebral end plates; 5. small, pointed, eroded odontoid with loss of cortex; 6. platybasia; 7. apophyseal joint erosion, blurred facets; 8. generalized osteoporosis of cervical spine; 9. a difference of 5 mm. or more between the posterior arch of atlas and the spinous process of axis from flexion to extension; and 10. secondary osteosclerosis of atlanto-axial-occipital area.

Diagnosis of rheumatoid arthritis was made if the roentgenogram disclosed 3 or more of these criteria. The severity of the changes increased with the duration of the disease. Those with advanced rheumatoid changes in the cervical spine showed definite changes in the hands and feet. Over two-thirds of the patients had positive latex fixation tests; however, rheumatoid arthritis in the cervical spine also occurred in patients who did not have the rheumatoid factor as measured by the latex fixation test. Subcutaneous nodules were present in 38.3 per cent of the cases.

The most common clinical manifestation of cervical spine involvement was severe and persistent pain. In extensive high cervical involvement the pain was referred to the occipital, temporal, and retroorbital areas.—*David Morse, M.D.*

Jacobs, Philip. Ankylosing spondylitis in children and adolescents. *Arch. Dis. Childhood*, Oct., 1963, *38*, 492–499. (From: Royal Orthopaedic Hospital, Birmingham, England.)

The author describes the occurrence of ankylosing spondylitis in 3 patients from 10 to 15 years of age. Three phases of the disease are apparent: 1) the prespondylitis syndrome with repeated attacks of pain and stiffness; 2) the florid phase of continuous symptoms and general illness; 3) a stage of declining activity and minor relapses.

Childhood cases are usually in the first phase and a relative latent period of several years may elapse before the second phase is reached, when a more rapid course may be seen. The early symptoms are varied and fleeting in nature and vary from a slight discomfort in the sacroiliac joint to moderate severe pain with actual limp. The clinical examination may reveal local tenderness to compression over the joints and limitation of abduction of the hip joints.

The author stresses the value of roentgenographic examination of the sacroiliac joints in early diagnosis. The most useful sign in a young patient is the presence of periarticular sclerosis. Later there are large subcortical erosions causing a widening of the joint space and patchy, mottled amorphous periarticular sclerosis. Fusion of the sacroiliac joints occurs late, as well as evidence of other joint involvement.—*James F. Martin, M.D.*

De Conti, Dirceu. (Curitiba, Brasil.) Osteoartritis septica de la cadera. (Septic osteoarthritis of the hip.) *Rev. ortop. y traumatol.* (Latino Americana), July, 1963, *8*, 123–128.

Before the advent of antibiotics the mortality of septic arthritis was about 10 per cent. The use of antibiotics has decreased the mortality considerably but local complications frequently persist. The majority of the cases are caused by resistant strains of *Staphylococcus aureus* which reached the involved joint by hematogenous dissemination.

Since pyogenic osteomyelitis usually starts in the metaphysis and spreads upward into the epiphysis, the joint is prevented by the physeal cartilaginous plate. A considerable portion of the metaphysis of the upper portion of the femur is within the articular capsule so that pyogenic arthritis is most common in the hip. In this series of 15 cases, 2 were bilateral.

The acute phase is usually manifested by a clinical picture of septicemia. The involved joint is swollen and the head of the femur may be displaced slightly laterally. Early treatment in the first 24 to 48 hours includes immobilization and aspiration or drainage. Delaying treatment leads to complications such as abscess formation, partial destruction of the head of the femur and various degrees of residual disability. Resection of sequestra and granulation tissue may be necessary. In this series of 15 patients there were 11 hips which eventually became ankylosed.

In another series of 14 patients treatment was conservative with traction, manipulation under anesthesia, and antibiotics. Ankylosis occurred in 6 hips. Three of these patients had persistent foci of active infection after 14, 21 and 53 months.—*Charles M. Nice, Jr., M.D., Ph.D.*

Bohatirchuk, F. Use of x-rays in the detection of calcium in biological tissues at microlevel. *Canad. M. A. J.*, Dec. 7, 1963, *89*, 1171–1177. (From: Department of Anatomy, Faculty of Medicine, University of Ottawa, Ottawa, Ontario, Canada.)

This article details methods of x-ray microscopy used in medico-biological research:

1. X-ray projection microscopy, where the section is placed close to the target of the x-ray tube so there is direct magnification.

2. Historadiography (or contact microradiography) where the section is placed in close contact with the film and the radiograph is then studied microscopically.

Although historadiography shows the presence of calcium, to obtain localization in specific tissue stain historadiography is used. There is no morphological differentiation between calcium carbonate and calcium phosphate by this method.

There are reproductions of 16 historadiographs and 2 macroradiographs which show the findings in osteomyelitis, Paget's disease, oil granuloma, osteoporosis and sarcoma as well as in growing rat bone.

Serial historadiographs provide the most accurate method of identifying calcium at the microlevel. The method can be further improved by the use of emulsion of finer grain and thinner cuts of undecalcified tissues which at present are 10μ thick.—*Martha Mottram, M.D.*

Blood and Lymph System

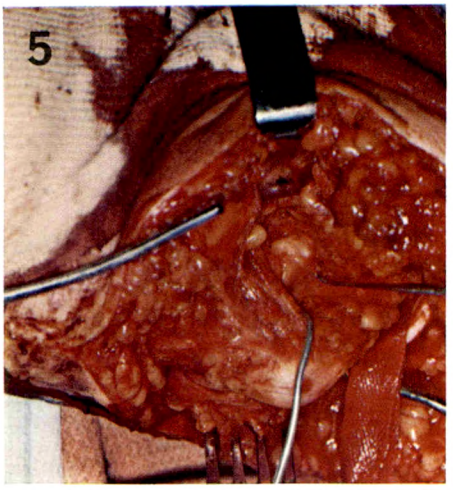
Whitaker, William. The clinical application of coronary arteriography. *Clin. Radiol.*, Oct., 1963, *14*, 397–401. (From: The General Infirmary at Leeds, England.)

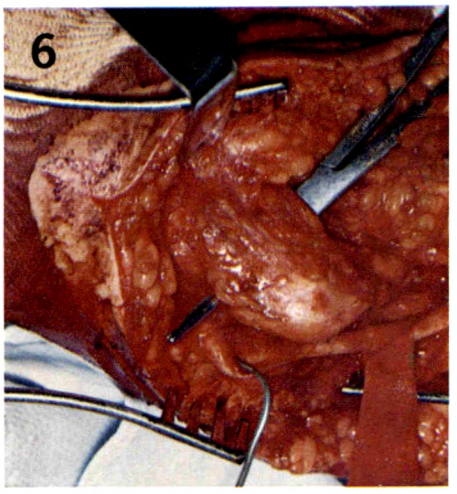
The author's purpose is to discuss the indications for coronary arteriography.

The best evidence for the diagnosis of coronary artery disease is provided by the history, and angina pectoris must be the clinical diagnosis in every patient presenting a typical story of chest pain. The mechanism producing the pain is myocardial anoxia and coronary artery disease is the commonest cause of this anoxia. It is in the group of patients with chest pain and negative electrocardiographic findings that coronary arteriography can provide valuable diagnostic information.

Coronary arteriography was performed by retrograde aortography in 42 patients and was technically successful in 33. In this group there were 16 with a classic history of angina pectoris. Of 10 with a normal electrocardiogram at rest and after exercise, there were 4 in which a coronary block was shown. Of 3 with normal electrocardiographic findings at rest and a positive exercise test, 2 had a coronary block. Three had inconclusive electrocardiograms, and in each a coronary block was demonstrated. Twelve of the 42 patients had chest pain of obscure origin and of these only 1 had a coronary block. Six of these 12 had normal electrocardiograms, 4 were inconclusive and 2 had abnormal tracings.

Chest pain may arise from the involvement of sensitive structures in the mediastinum by various pathologic processes or may be referred from abdominal disease. Conditions such as ischemic heart disease, valvular heart disease, spontaneous pneumothorax, dissecting aneurysm of the aorta, cholecystitis, hiatus hernia, and other esophageal lesions may cause pain simulating that of angina pectoris. In these cases where surgery is contemplated coronary arteriography may be a helpful preliminary procedure.—*Samuel G. Henderson, M.D.*







POSTPOLIO PARALYSIS WITH A FLAIL RIGHT SHOULDER AND ELBOW *(CONTINUED)*

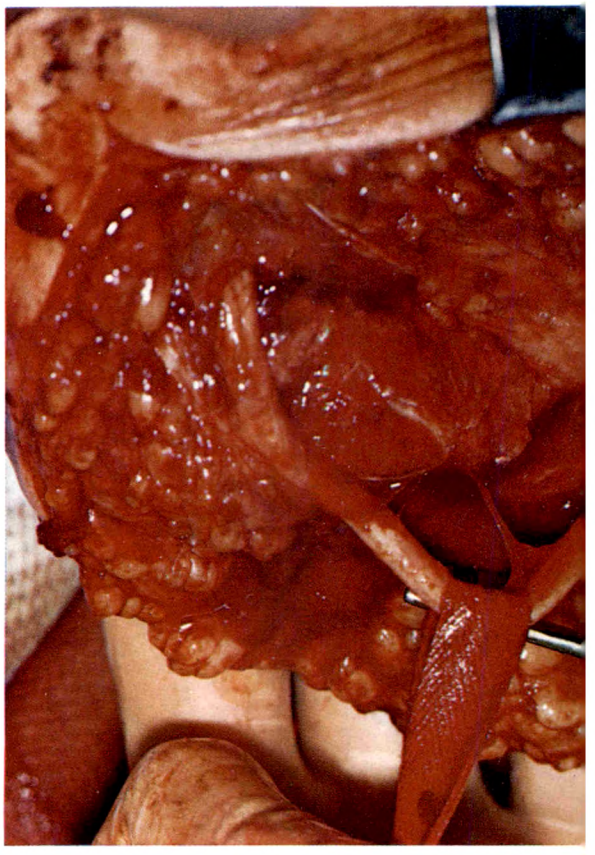
Mobilization of common flexor muscle mass arising from the medial epicondyle.

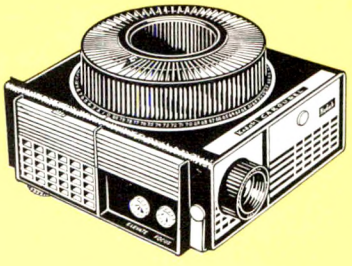
Figure 5: Demonstration of median nerve.

Figure 6: Freeing of flexor muscle mass from underlying attachment while median and ulnar nerves are protected.

Figure 7: Osteotomy of medial epicondyle. The attached flexor muscle mass has been mobilized and reflected distally for photograph.

Figure 8: Medial epicondyle with flexor muscle mass reattached 5cm more proximally on the humerus by a wire suture.





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SURGERY:

Steindler flexor plasty operation to restore ability to flex right elbow



Figure 3: 15cm incision over medial aspect of right elbow.

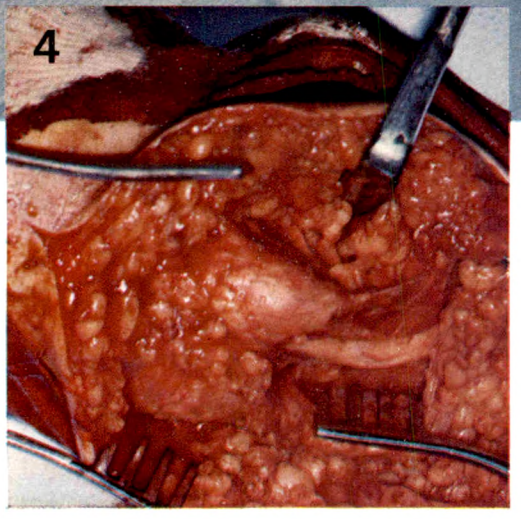


Figure 4: Thick subcutaneous layer has been dissected away to show medial epicondyle and the ulnar nerve immediately posterior to it.

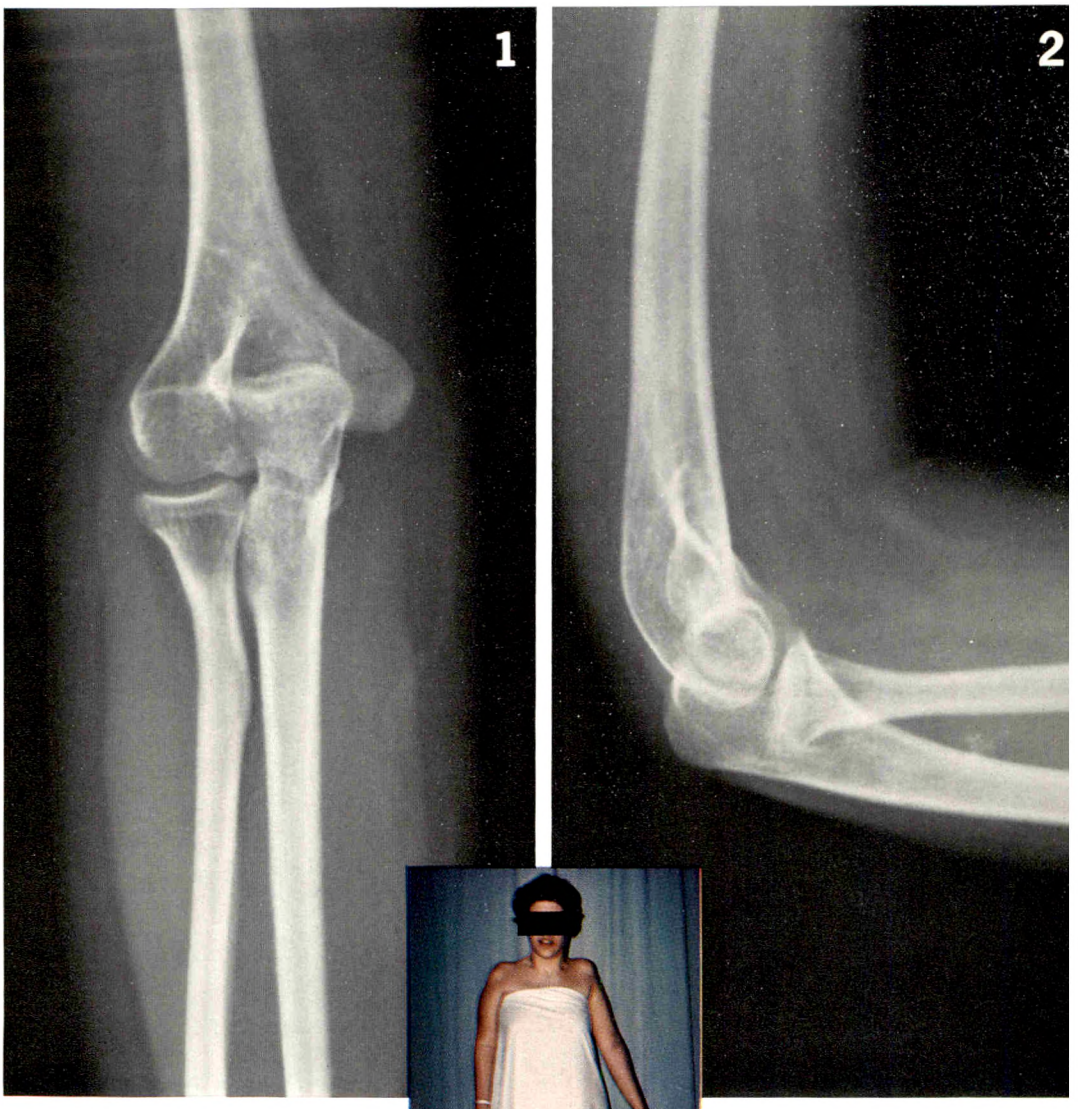
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POSTPOLIO PARALYSIS WITH A FLAIL RIGHT SHOULDER AND ELBOW

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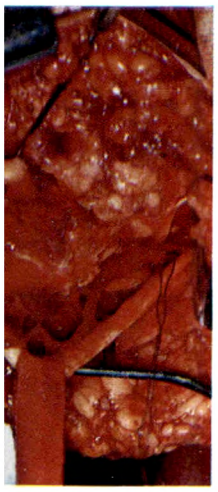
Postpolio paralysis with flail right shoulder and elbow



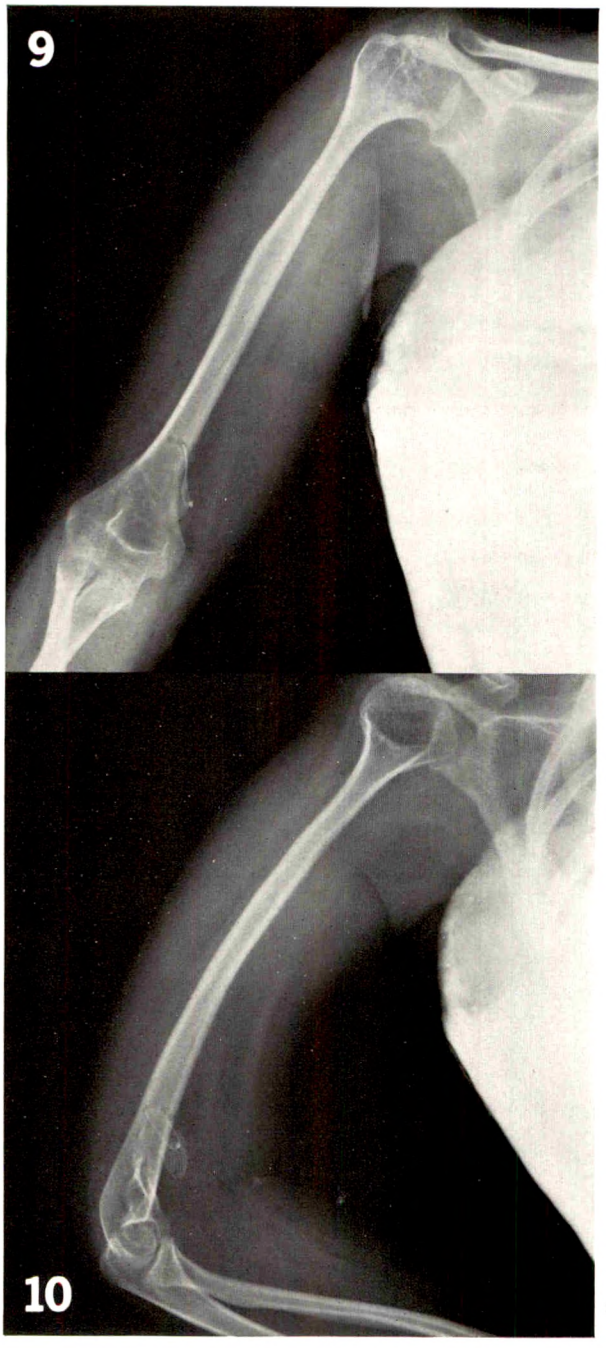
Figures 1 and 2: Preoperative anteroposterior and lateral radiographs of the right elbow. Elbow passively flexed for the lateral radiograph. Note normal bony structure.



Photographs: Front and side views of patient showing inability to flex right elbow.







Figures 9 and 10: Postoperative antero-posterior and lateral radiographs of the right elbow. Medial epicondyle is seen transplanted proximally and held by wire suture.

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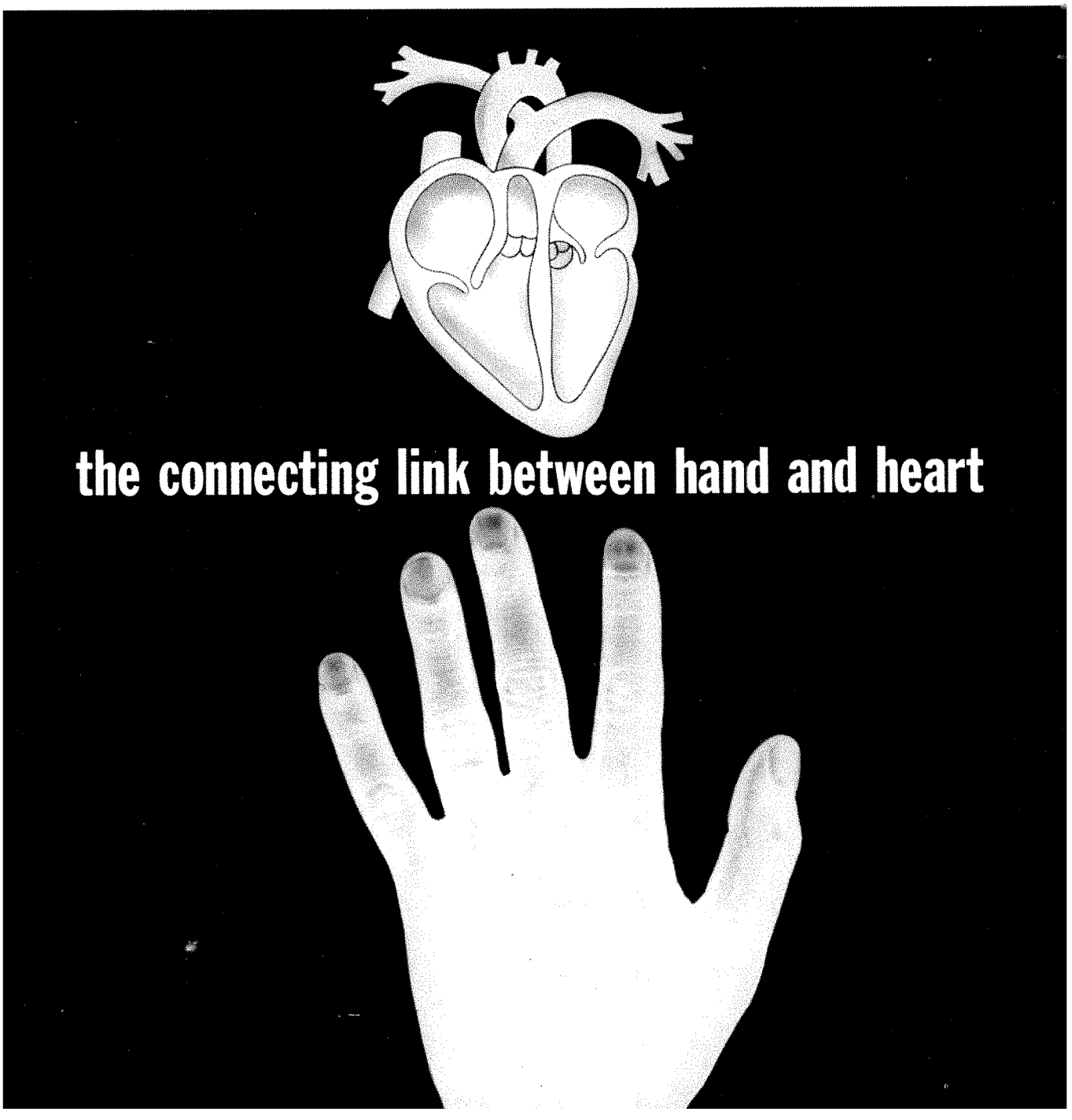
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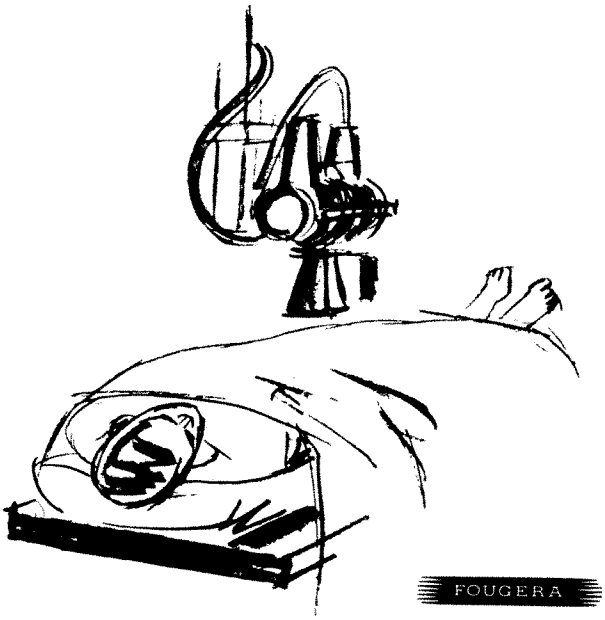
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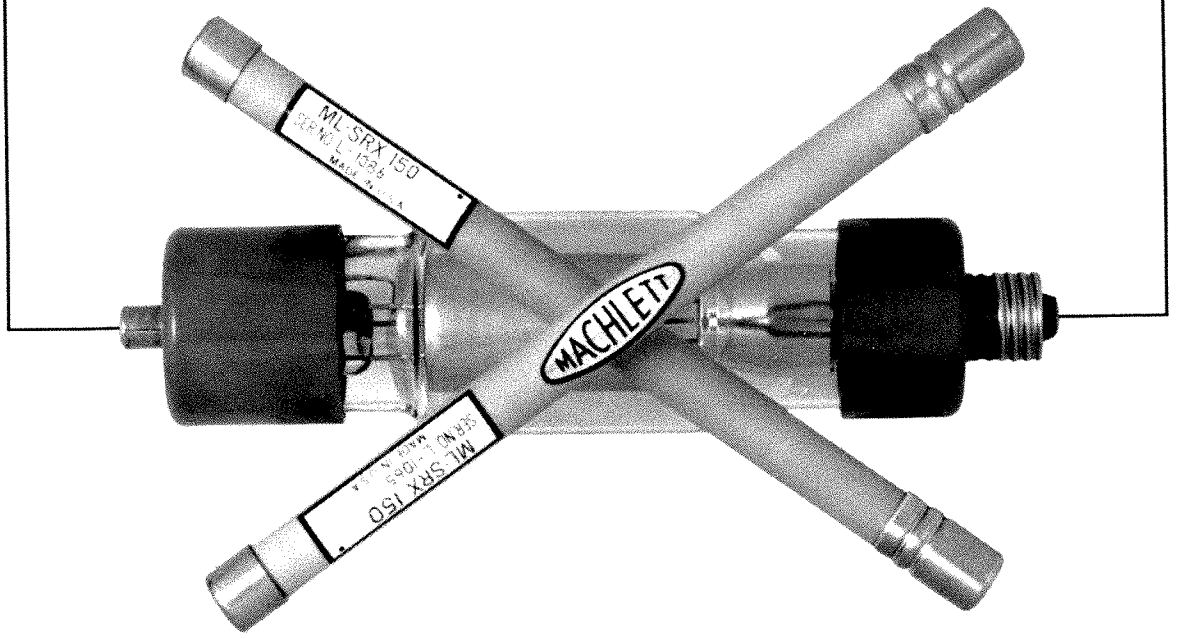
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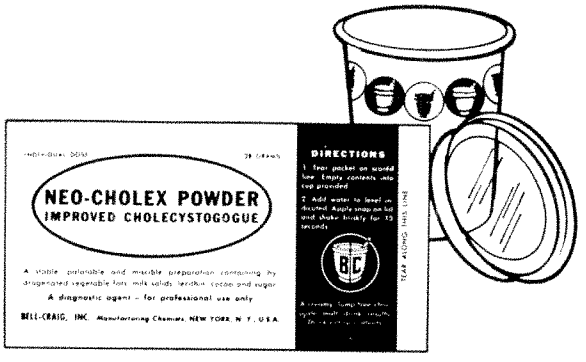
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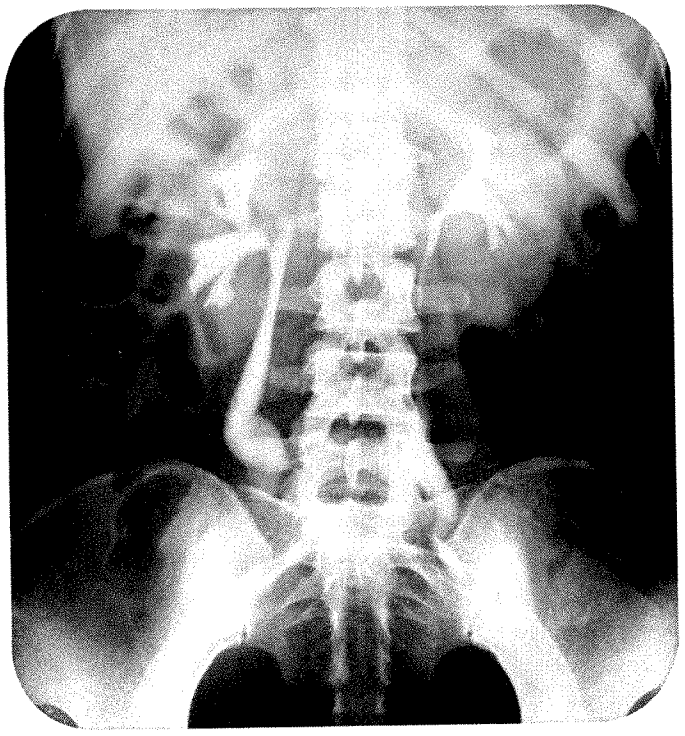
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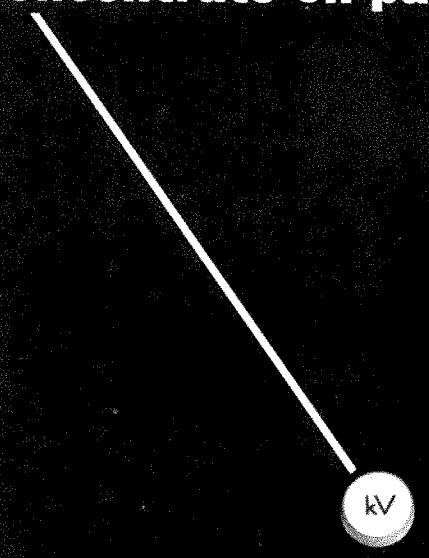
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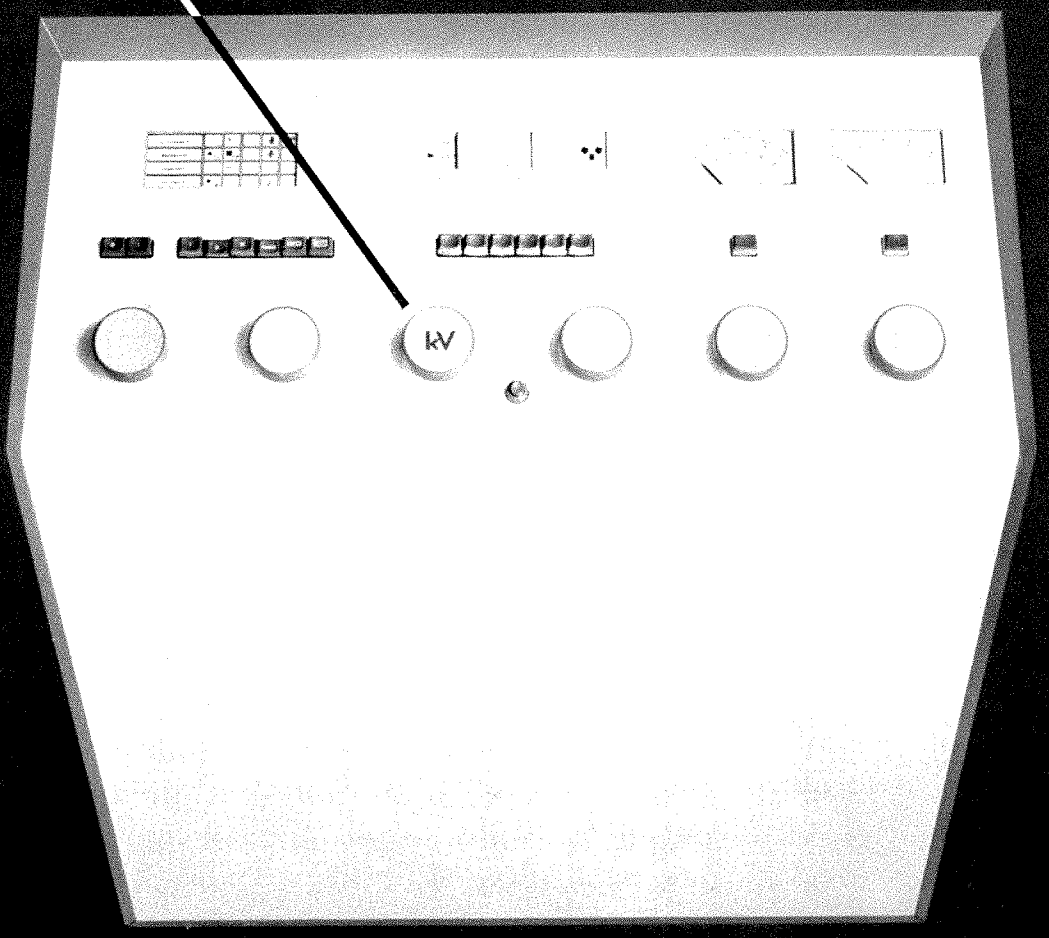


kV is the only factor your technician is required to select. Once that is done, she presses the exposure button and the automatic system does the rest.

Proper density is guaranteed by the Amplimat, the automatic ionization-type exposure control which measures the average X-ray quantum acting on the film over several selectable areas. To ensure positive positioning, the lightbeam indicator projects the location of the measuring areas on the patient. Just as in the case of the modern camera, automatic exposure with Amplimat pays off in consistent production of optimum radiographs.

Predictable contrast is guaranteed by

Only Norelco offers this system, which provides flawless radiographs automatically



MANUFACTURED FOR NORELCO BY PHILIPS MULLER

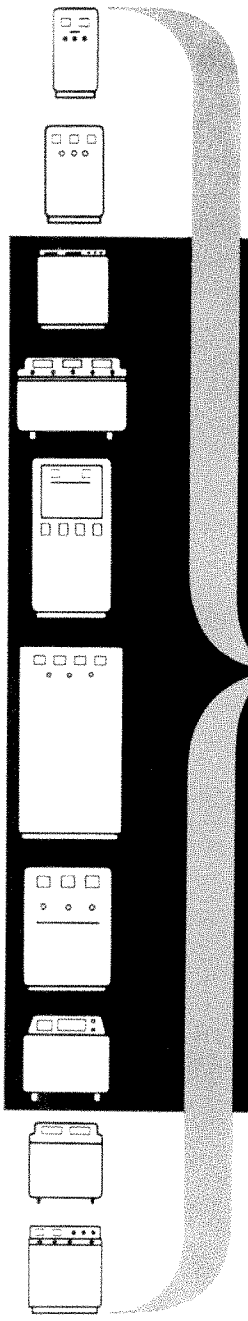
automatic line correction and by the essentially constant voltage output characteristic of a 3-phase generator.

Sharpness is increased by the 'continuously falling tube-load principle', which permits shortest possible exposure time with maximum available tube loading. Optimum sharpness is assured when taking advantage of the high load characteristic and small focal spot sizes of the Super Rotalix tube.

Our systems engineers are available, at your convenience, to discuss this system.

North American Philips Company, Inc., *Medical Division*, 100 East 42nd Street, New York, New York 10017. In the Chicago area: 749 Howard Street, Evanston, Illinois.

Norelco®



SIEMENS

Compatibility
Dependability



Film
after film
of
consistent
density

IONTOMAT

If you are not satisfied with your present phototimer system or if you are not using automatic timing, perhaps now is the time to investigate a unique ionization-chamber controlled system, time-proven, and trouble free.

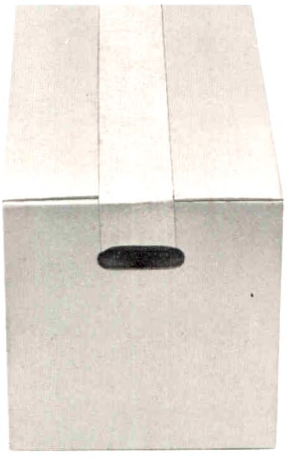
The IONTOMAT is compatible with any make of generator.

SIEMENS MEDICAL OF AMERICA INC.
685 LIBERTY AVE. · UNION · N. J.

gaf Ansco

LIQUAMAT®

Chemicals





for automatic
X-ray film
processors



now come in
color-coded
square plastic bottles
for easier handling
and storage and . . .

. . . greater convenience. And that's not all!



Handy new color-coded one-gallon plastic containers let you choose the chemical you wish at a glance, help prevent errors even when you're rushed. (Developer—red; Fixer—blue.)



Containers have square sides . . . making them far easier to store and handle. And they're real lightweights, too. So convenient, you'll be glad we've designed them to be reusable!

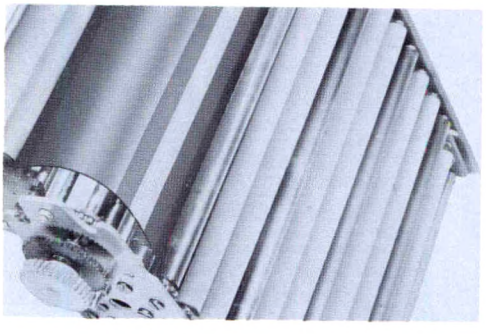


= 5 gal. working solution

There are fewer separate Liquamat chemicals than in any similar "package" on the market. And they come in gallon bottles, so you can work with five gallon "multiples" of solution.

AUGUST
SEPTEMBER
OCTOBER
NOVEMBER
DECEMBER
JANUARY

Liquamat solutions have unusually long life . . . to process 60,000 or more films, or give you five to six months active service (with the recommended use of Liquamat replenishers).



And Liquamat chemicals are the cleanest you can use . . . free from oxidation and sludging. They virtually eliminate all processing artifacts, staining, and minimize roller build-up.

Add up the features and look at the benefits you get: Greater convenience; cleaner working; longer life; improved radiographic quality. All important reasons why it will pay you to start using Ansco Liquamat chemicals in your automatic processor. Call your GAF-Ansco Technical Representative . . . and start today!

gaf Ansco
X-RAY PRODUCTS
GENERAL ANILINE & FILM CORPORATION
BINGHAMTON, NEW YORK

Printed in U.S.A.

CLYSODRAST® FOR EFFECTIVE CLEANSING OF THE COLON

CLYSODRAST used as an enema is an effective means of cleansing and preparing the colon for radiologic examination.

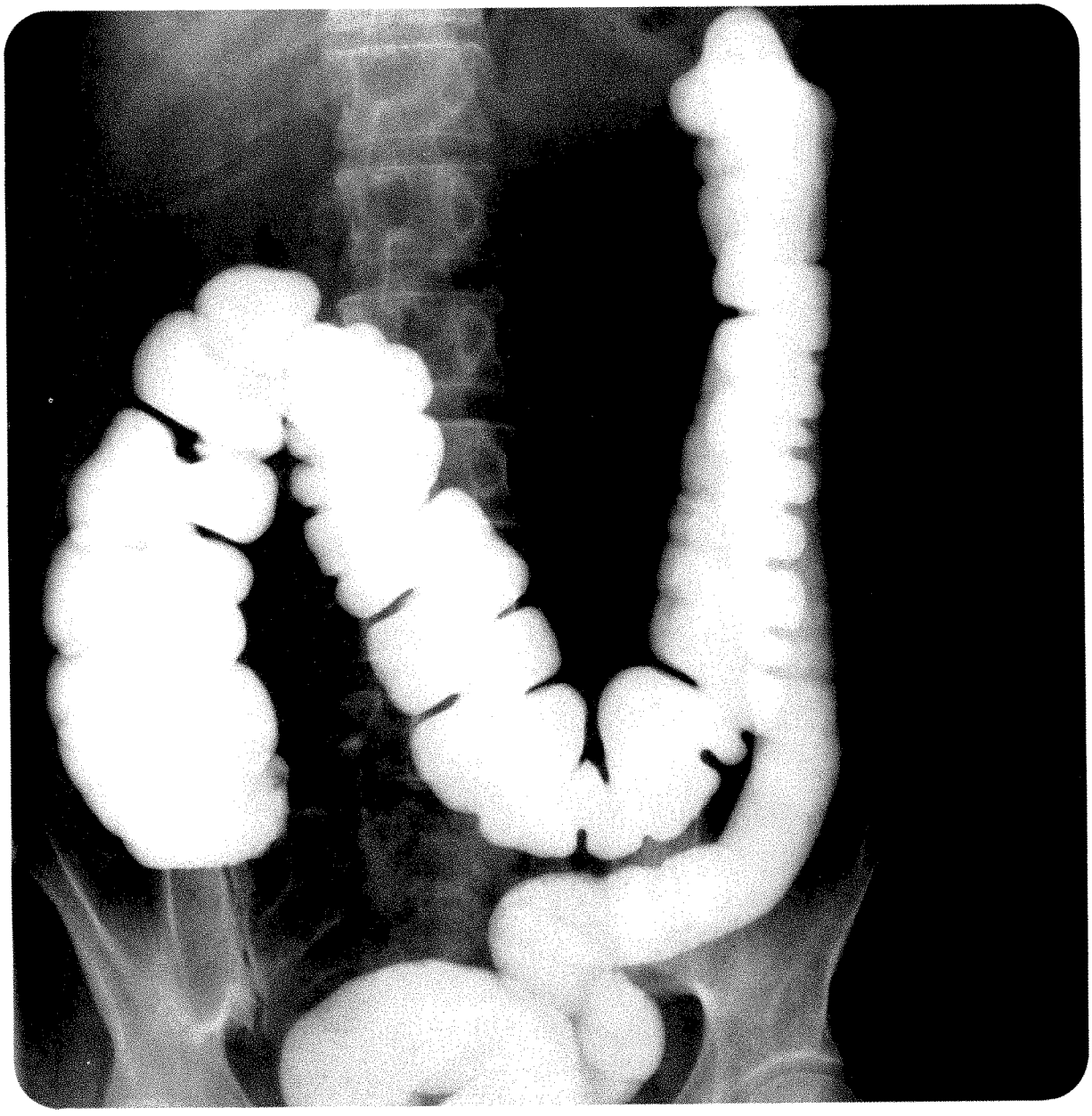
The effective agent in CLYSODRAST is a nonirritant which stimulates the musculature to contract. The Tannic Acid in CLYSODRAST has a favorable influence on the deposition of Barium Sulfate on the mucosa and aids in preparation of the colon.

CLYSODRAST: each individual dosage pack contains 1.5 mg. of 4,4'-(DIACETOXYDIPHENYL)-(PYRIDYL-2)-METHANE AND 2.5 Gms. of TANNIC ACID N.F., in a readily soluble form.

Caution: Cramping, weakness, nausea and fainting occur more often with this product than with less effective enemas. It should be employed with care where colitis is known to exist.
Warning: Use with great care in patients where old age, debility, cardiovascular or other diseases are present.

BARNES-HIND BARIUM PRODUCTS
DIVISION OF BARNES-HIND PHARMACEUTICALS, INC.
Sunnyvale, California

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When

the clinical situation demands
conclusive, differential diagnosis
consider these contrast media

Hytrast® (iopydone, iopydol)
Ethiodol® (ethiodized oil)
Lipiodol® 28% (iodized oil)
Lipiodol® 40% (iodized oil)
Ascendant Lipiodol® (floating iodized oil)
Visciodol® (Lipiodol-sulfanilamide)

...and procedural aids

Colo-Bar® (ready-to-eat cholagogue)
Polysorb® Hydrate (for radiation dry skin)

CONTRAST MEDIA — DEVELOPMENTS OF GUERBET LABORATORIES

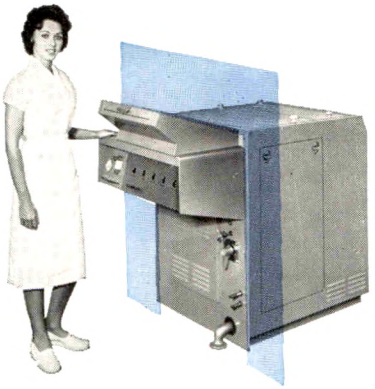
FOUGERA **E. FOUGERA & COMPANY, INC.** HICKSVILLE, L. I., NEW YORK

advances
you'll find in new PICKER
PIXAMATIC film processor
with VIBRAFEED Autoloader



6 minutes
dry to dry

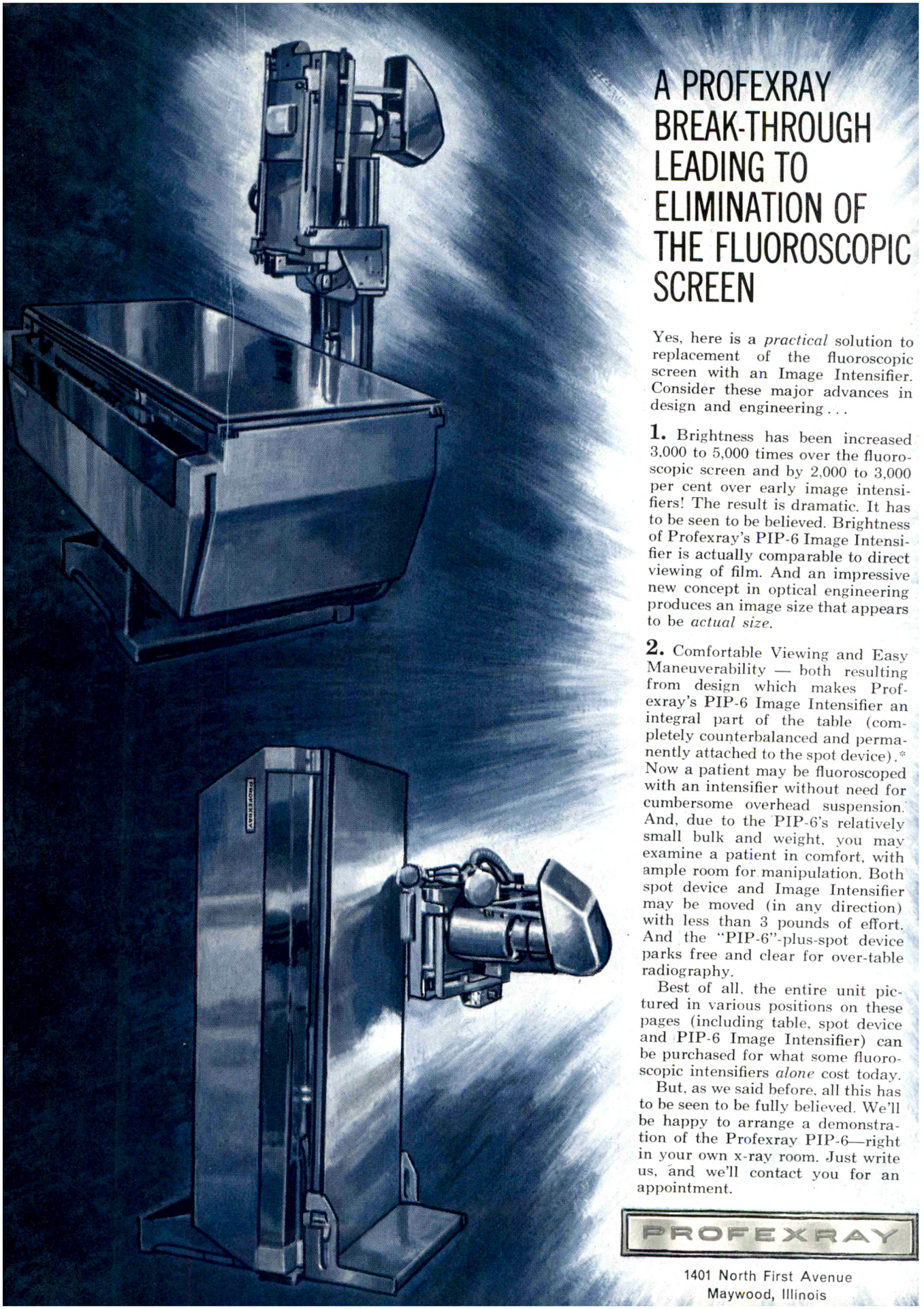
- **115 FILMS PER HOUR** *(average assortment)*
- **NO DAILY CLEANING** *(total wetting precludes "crossover crusting")*
- **HANDLES INCOMING WATER UP TO 80°** *without need for cooler*
- **PUMPLESS REPLENISHMENT** *(positive-metered Venturi flow)*
- **WORKS IN BRIGHT LIGHT** *(needs dark only during loading)*
- **INTERCHANGEABLE RACK UNITS** *(easily lifted from waist-high cabinet)*



takes very little floor space . . .
36″ x 30″ in the lightroom side
19″ above-floor extension into
darkroom for thru-the-wall Autoloader

Picker x-ray

PIXAMATIC is chockfull of new ideas in film processing equipment: let your Picker representative fill you in on the full story or write



A PROFEXRAY BREAK-THROUGH LEADING TO ELIMINATION OF THE FLUOROSCOPIC SCREEN

Yes, here is a *practical* solution to replacement of the fluoroscopic screen with an Image Intensifier. Consider these major advances in design and engineering . . .

1. Brightness has been increased 3,000 to 5,000 times over the fluoroscopic screen and by 2,000 to 3,000 per cent over early image intensifiers! The result is dramatic. It has to be seen to be believed. Brightness of Profexray's PIP-6 Image Intensifier is actually comparable to direct viewing of film. And an impressive new concept in optical engineering produces an image size that appears to be *actual size.*

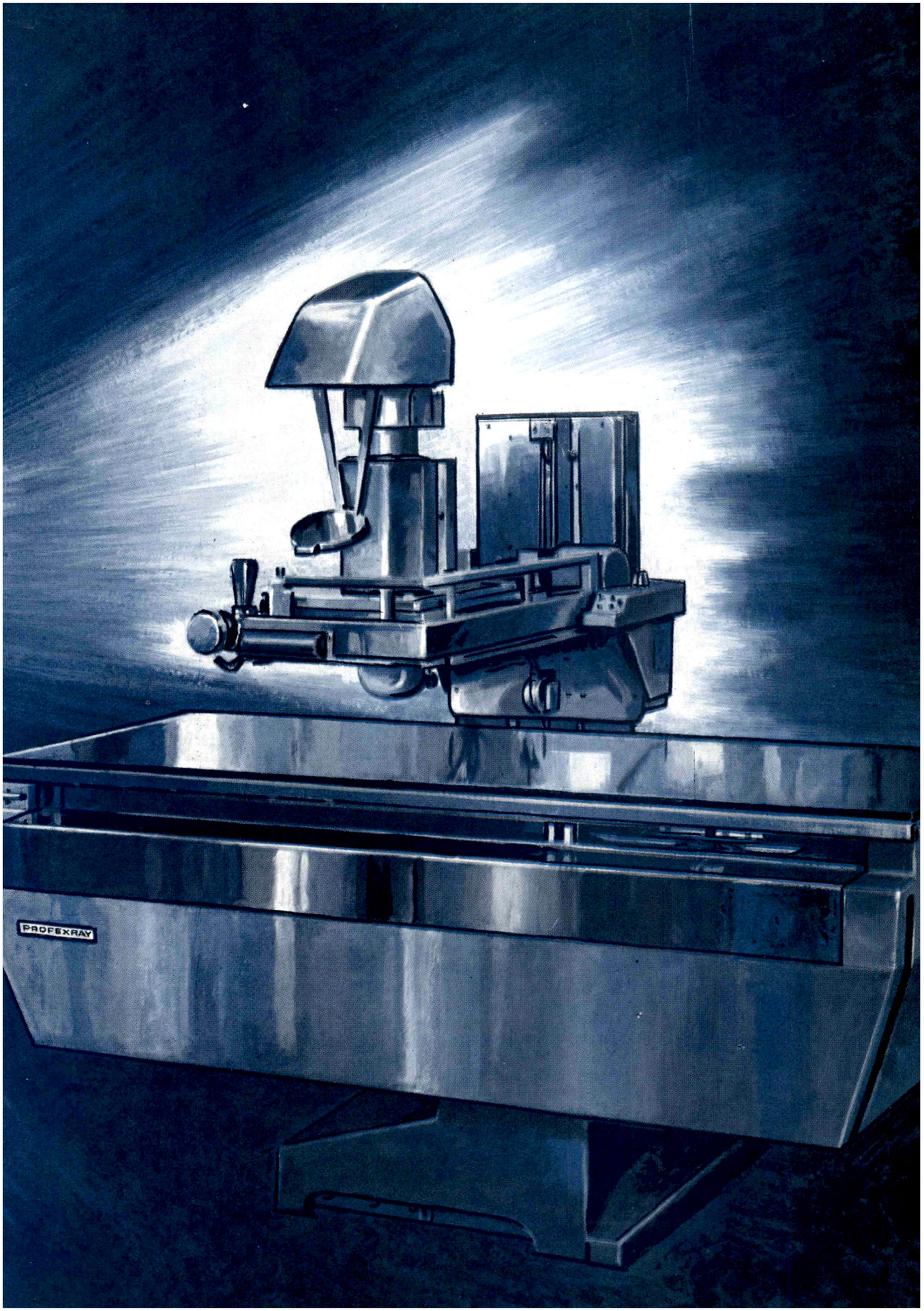
2. Comfortable Viewing and Easy Maneuverability — both resulting from design which makes Profexray's PIP-6 Image Intensifier an integral part of the table (completely counterbalanced and permanently attached to the spot device).* Now a patient may be fluoroscoped with an intensifier without need for cumbersome overhead suspension. And, due to the PIP-6's relatively small bulk and weight, you may examine a patient in comfort, with ample room for manipulation. Both spot device and Image Intensifier may be moved (in any direction) with less than 3 pounds of effort. And the "PIP-6"-plus-spot device parks free and clear for over-table radiography.

Best of all, the entire unit pictured in various positions on these pages (including table, spot device and PIP-6 Image Intensifier) can be purchased for what some fluoroscopic intensifiers *alone* cost today.

But, as we said before, all this has to be seen to be fully believed. We'll be happy to arrange a demonstration of the Profexray PIP-6—right in your own x-ray room. Just write us, and we'll contact you for an appointment.

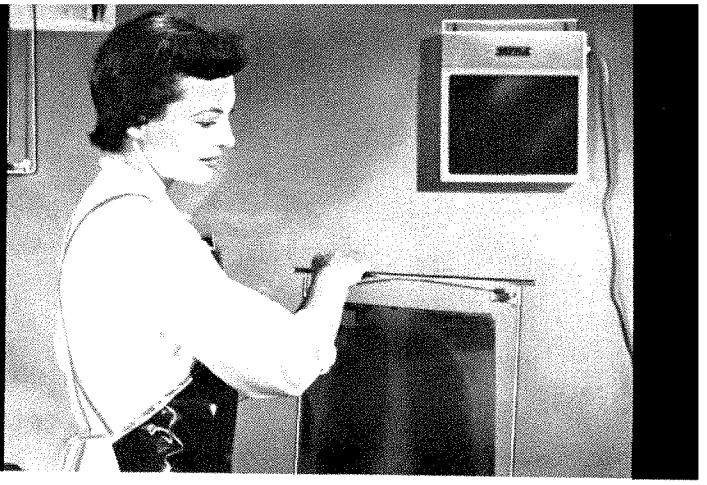
PROFEXRAY

1401 North First Avenue
Maywood, Illinois


PROFEXRAY

Halsey Safelights

14 different models to choose from...





No. 275-SL-S

COMBOLITE DELUXE

Stainless steel — only 3¼" deep

Compact combination of 8 x 10" safelight and 4 x 10" wet-film viewer — with built-in safeguard to prevent accidental lighting of viewer section. **No. 275-SL-S**

COMBOLITE

Same as above, but fabricated of cold-rolled steel with baked-on Silvergray finish. **No. 275-SL**

MONOLITE DELUXE

Stainless steel — only 3¼" deep

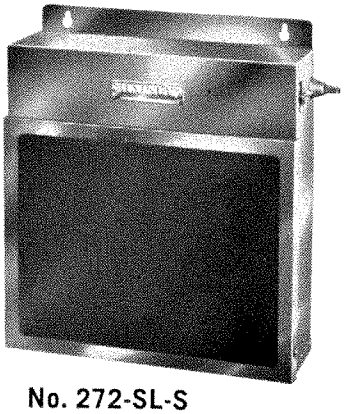
Extra-thin 8 x 10" safelight . . . valuable in tight quarters; can be mounted over the developing tank, yet will not be in the way of film hangers. **No. 272-SL-S**

MONOLITE

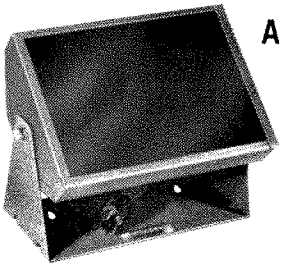
Same as above, but with steel housing finished in baked-on Silvergray. **No. 272-SL**

MONOLITE JR.

A 5 x 7" version of the Monolite. **No. 279-SL**



No. 272-SL-S



ADJUSTABLE SAFELIGHT

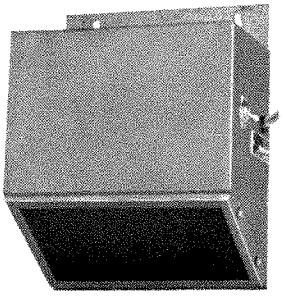
Tilts to any desired angle to throw light right where it is needed.

No. 274-SL

ANGL-LITE

An economical angulated safelight designed to provide a good spread of light over the bench or tank.

No. 276-SL



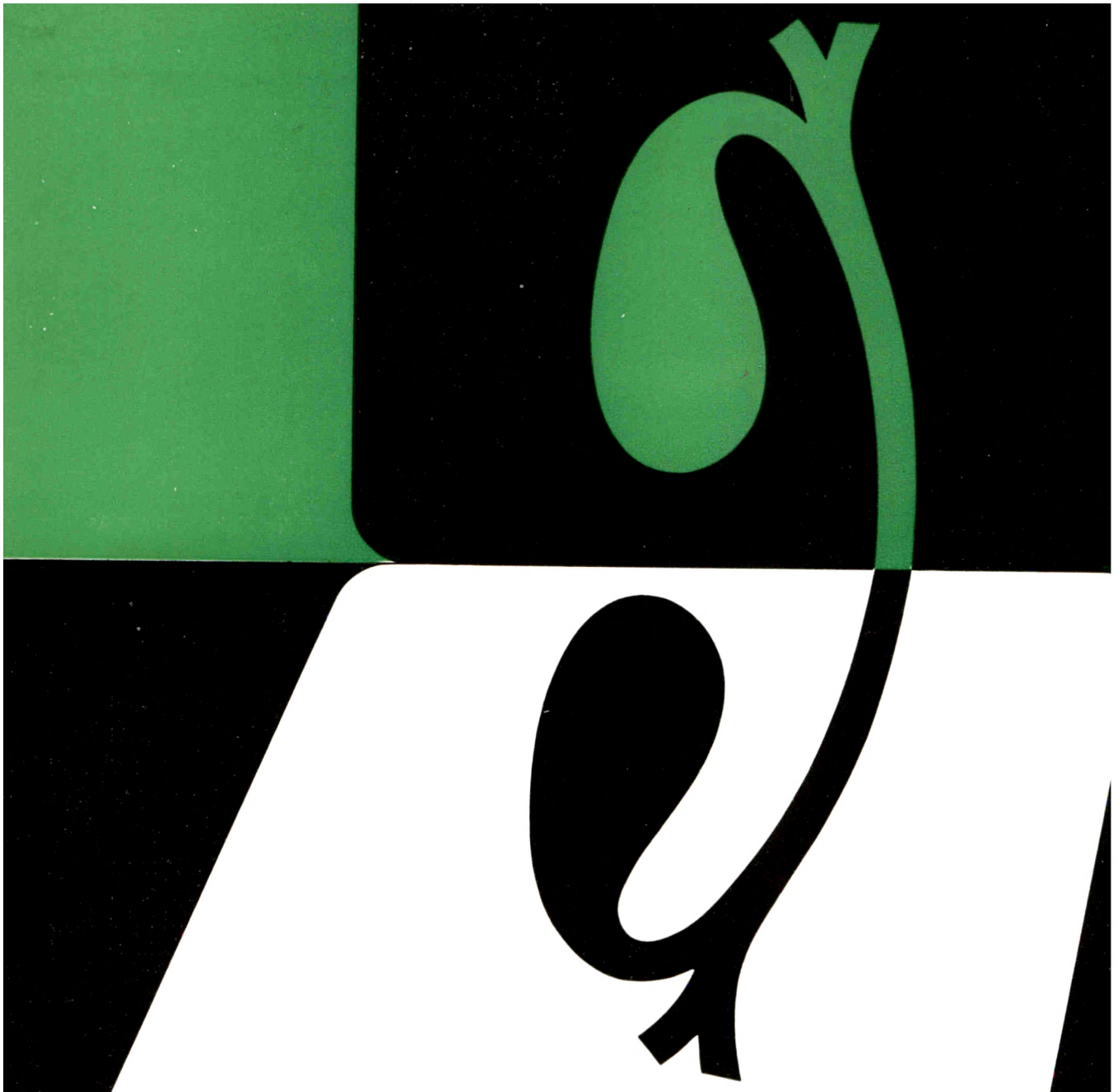
Also: other combination safelight-viewer models; circular safelights; and 10 x 12" wall and ceiling hung models.

Through your dealer
Write for detailed literature

HALSEY

HALSEY X-RAY PRODUCTS, INC.
1425-37th Street, Brooklyn 18, N.Y.

CHICAGO: 7321 Monticello Ave., Skokie, Illinois



In cholecystography coming events will cast their shadows

The need for gallbladder surgery, for example. *Better contrast visualization*, of course means more *precise diagnosis* — the reason why well-tolerated Telepaque (brand of iopanoic acid) has so firmly established itself in oral cholecystography and cholangiography. Telepaque not only provides gallbladder shadows with better contrast, and improved visualization of the ducts, but it does so without disturbing gallbladder physiology. Convenient and economical, too.

Side effects, which are usually mild, include nausea, vomiting, diarrhea, dysuria. *Contraindications* are acute nephritis and uremia, and gastrointestinal disorders which prevent absorption of the medium.

Usual Dosage: 3 Gm. (6 tablets) at night after a light supper. Tablets of 500 mg., envelopes of 6 tablets, boxes of 5 and 25 envelopes; and bottles of 500.

well-tolerated medium with distinctly better contrast **Telepaque®**

brand of iopanoic acid

Winthrop WINTHROP LABORATORIES



Added insight to help speed surgical recovery... fast, accurate blood volume measurements

In 15 minutes, VOLĒMETRON measures blood volume accurately to within 100 ml for an adult, 20 ml for a child. VOLĒMETRON can provide this potentially lifesaving information at the bedside preoperatively to establish a baseline for replacement therapy...in the operating room to monitor blood loss...in the recovery room to detect concealed hemorrhage and avoid undertransfusion, overtransfusion (or unnecessary transfusion). Accurate blood volume information, with hematocrit data, also aids in diagnosis and management. For example, conditions characterized by variations in red cell volume, plasma volume, total blood volume,.and fluid translocation and loss can be more closely controlled with precision measurements. VOLĒMETRON is attested by over 30 clinical papers. This versatile, uniquely-warranteed instrument is also used for other diagnostic applications such as the T-3 and Schilling Tests. For further information and literature, or to arrange a demonstration of VOLĒMETRON in your hospital, contact Ames Atomium, Inc., 575 Middlesex Turnpike, Billerica, Massachusetts 01865.

73364



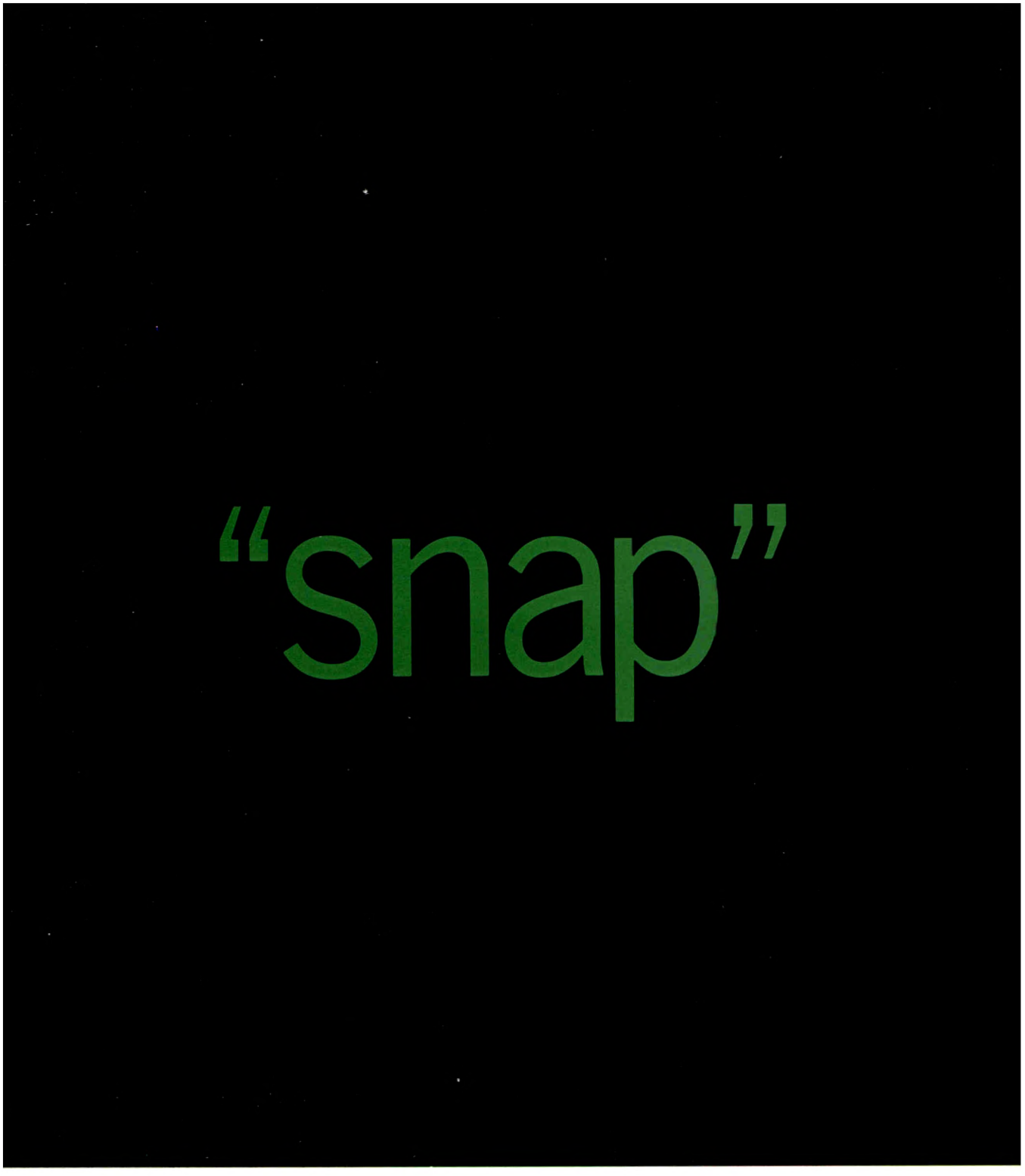
VOLĒMETRON®

Original Automatic Electronic Blood Volume Computer

ames atomium, inc.

Billerica, Massachusetts

affiliated with Ames Company, Inc.



What new film base speaks for itself?

CRONAR® (snap) polyester film base

Snap a sheet of CRONAR* film. Notice the crisp, distinctive sound. This is a completely new material as different from ordinary acetate as acetate was from glass plates. For CRONAR is radically different.

This film goes up on the viewbox with a decisive snap, and the crisp material is virtually untearable. These handling qualities are clearly paralleled in the sharper, brighter image you read.

The base lets more light through, because it's a thinner base; it brings front and back emulsion layers closer together to reduce parallax, to give you sharper images.

CRONAR is chemically inert and free of radioactive contamination – significantly reducing the incidence of bothersome black spots.

CRONAR is almost impervious to water and processing solutions, too. So it won't go limp in automatic processing machines. Result: CRONAR film transports better. Three major hospitals where processors jammed several times a week switched to CRONAR – and haven't had a film jam in a year and a half.

Ask your Du Pont Technical Representative to show you the new look in Du Pont films for radiology – CRONAR – the look you can hear.

See Du Pont Show, New York World's Fair, '64-'65

*Du Pont's registered trademark for its polyester film base

PHOTO PRODUCTS DEPARTMENT

DU PONT
REG. U. S. PAT. OFF.

BETTER THINGS FOR BETTER LIVING
. . . THROUGH CHEMISTRY





No matter how good your scanner may be, it's no good to you at all if it's out of commission. Your busy schedule simply can't tolerate the disruptions growing out of equipment "downtime".

Across the United States, there are over 400 Picker-employed servicemen working out of 112 Service Centers and backed up by over 40 service specialists. This service force is substantially larger than that of all other nuclear instrument manufacturers *put together.*

PICKER nuclear

Magna Scanner

the *versatile* scanner / the *proven* scanner

PICKER NUCLEAR
DIVISION / PICKER X-RAY CORPORATION
WHITE PLAINS, NEW YORK

the ULTIMATE in table design!

Standard ULTIMA-105

HEAVY DUTY
RADIOGRAPHIC-FLUOROSCOPIC

X-RAY TABLES

FOR THE PATIENT:
the ultimate in comfort

FOR THE RADIOLOGIST: the ultimate in efficiency and convenience... less work and less effort to do it!

FUNCTIONAL CONVENIENCE: Maximum accessibility to the table. There's NO FRONT LEG, covered up or exposed, to keep the radiologist away from the table and spot film device. He can work comfortably right up against the inward slope of the table body (just 14 inches away from the center) without back-breaking bending and strain, resulting in more ease and much less effort.

• Choice of manual, semi-automatic or fully automatic Spot Film Device. It is quickly, easily "parked" out of the way, leaving table top free and clear. Movement is smooth and light because of the weight-saving counterbalance method. Low profile of tower and spot film device in "parked" position eliminates interference with shock-proof radiographic cables.

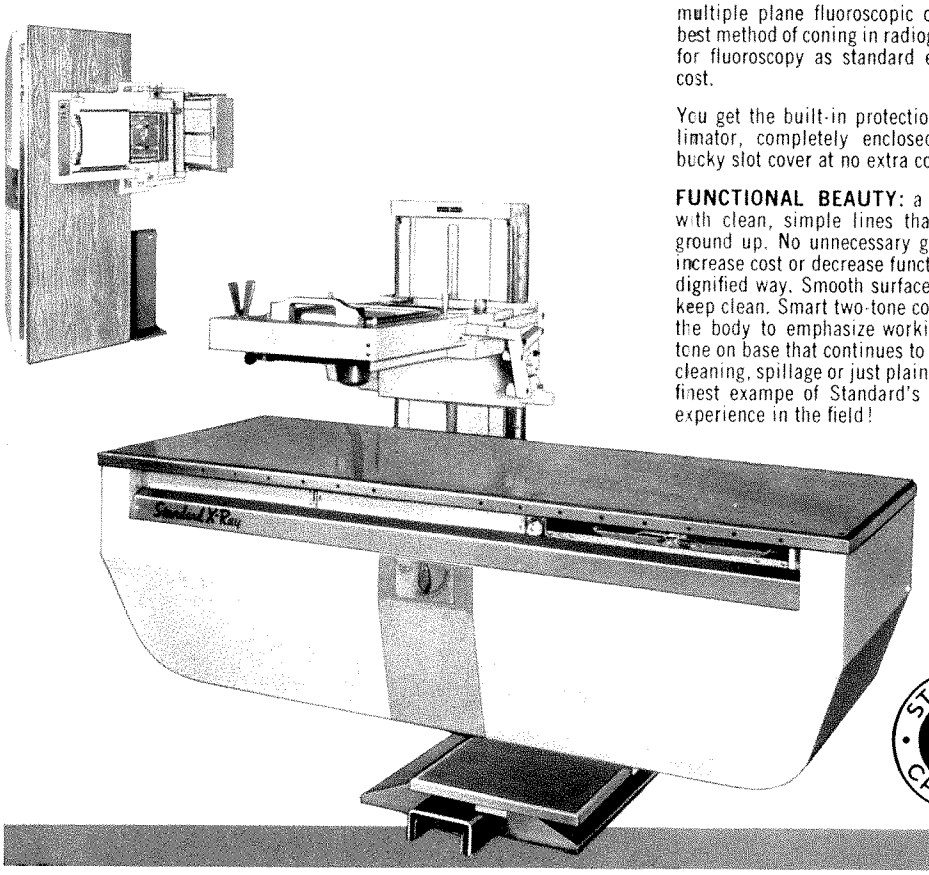
• Table operates on a single fulcrum. Tilts 90° vertical, and full 15° Trandelenburg. Automatic horizontal stop is standard equipment. Provides coast-free tilting from foot switch on base or hand switch on spot device. A retractable step for patient's convenience is standard. Features power driven moving table top (optional).

• Magnetic locks for all movements of the fluoroscopic and spot film staging. Switch control at operator's fingertips or automatic operation by the spot device action.

FUNCTIONAL EFFICIENCY: The manually operated multiple plane fluoroscopic collimator is the proven best method of coning in radiography. It is now adapted for fluoroscopy as standard equipment, at no extra cost.

You get the built-in protection of a fluoroscopic collimator, completely enclosed body and automatic bucky slot cover at no extra cost.

FUNCTIONAL BEAUTY: a truly handsome table with clean, simple lines that is all new from the ground up. No unnecessary gadgets or decoration to increase cost or decrease function. It is handsome in a dignified way. Smooth surfaces are easy to clean and keep clean. Smart two-tone colors: light sand-tone on the body to emphasize working area; dark hammertone on base that continues to look good after years of cleaning, spillage or just plain abuse. This table is the finest exampe of Standard's more than 50 years of experience in the field!



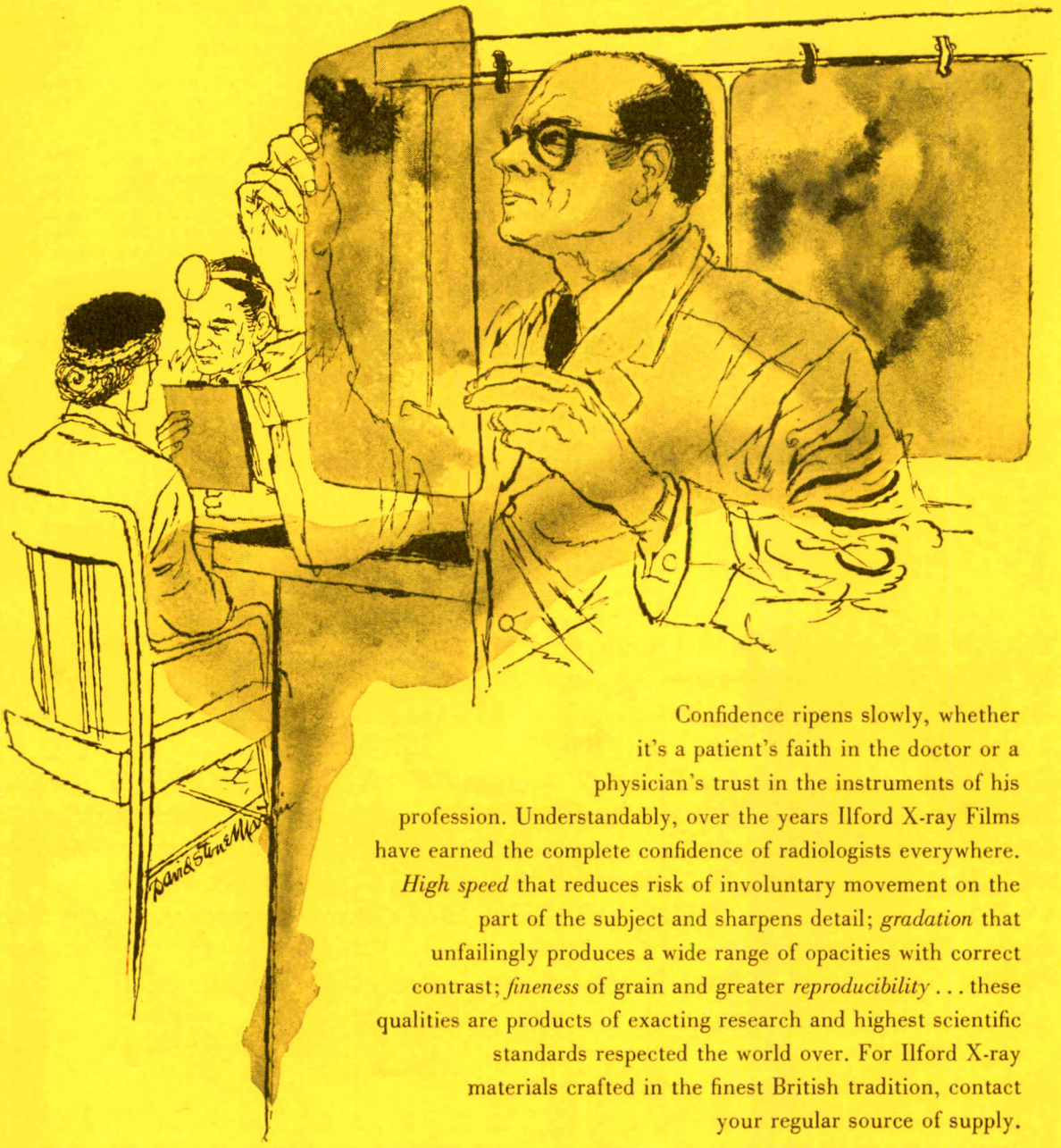
STANDARD X-RAY CO. CHICAGO

• See your "Standard" Dealer or write for Descriptive Literature.

Standard X-RAY COMPANY

1932 N. Burling St., Chicago 14, Ill.

Confidence



Confidence ripens slowly, whether it's a patient's faith in the doctor or a physician's trust in the instruments of his profession. Understandably, over the years Ilford X-ray Films have earned the complete confidence of radiologists everywhere. *High speed* that reduces risk of involuntary movement on the part of the subject and sharpens detail; *gradation* that unfailingly produces a wide range of opacities with correct contrast; *fineness* of grain and greater *reproducibility* . . . these qualities are products of exacting research and highest scientific standards respected the world over. For Ilford X-ray materials crafted in the finest British tradition, contact your regular source of supply.

ILFORD RED SEAL STANDARD ILFEX X-RAY FILMS • CHEMICALS • INTENSIFYING SCREENS

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HAND IN
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RADIOLOG
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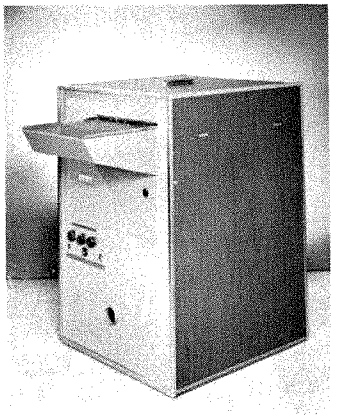
What's in a name? In the case of Philips, the name means many t
to many people. To the average consumer, Philips means, simply: lig
fine radios, outstanding household appliances..... almost everything
trical.
But to the world of medicine, Philips -- a world-embracing elect
industry -- means much more: For Philips have served generation
generation of radiologists. Always in the forefront in the developm
advanced equipment, Philips provide the radiologist with efficient
-- Image intensifiers, X-ray television and cinematography, diagr
dosimeters. From the most simple apparatus to the most con
Philips equipment is time-tested, time-honored for dependability.

PHILIPS

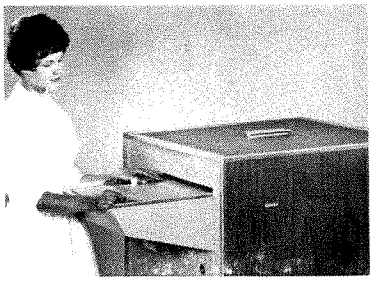
PHILIPS in the service of the medical

X-ray equipment for diagnosis and therapy X-ray tubes and X-ray accessories Dental equ
Electromedical and general hospital equipment Radiation measuring equipment Darkroom eq
SPECIALITIES: Image Intensification · Cineradiographic installations · X-ray television

For further information please apply to the Philips organisation in your Country. Sold in U.S.A. by: North American Philips Company, Inc., Medical & Dental Division, 100 East 42nd Street, New York 17, N.Y.



Why you should have a KODAK X-OMAT Processor, Model M4A



The Model M4A X-OMAT Processor is really compact. It needs less than half the floor area covered by a hospital bed.

Here are some of the benefits you'll have when the KODAK X-OMAT Processing System goes to work.

Heritage. For more than 7 years, KODAK X-OMAT Processors have been in action every day. At work throughout the world, in hospitals, clinics, and private radiological offices, they've processed more than 500,000,000 radiographs. Today's Model M4A X-OMAT Processor is the product of that experience.

Reliability. The X-OMAT system is the combined result of Kodak film-making knowledge and competence, chemical research, engineering skill. KODAK X-OMAT Processors are made by people who know the importance of the product they are producing. There is Kodak dependability in every unit.

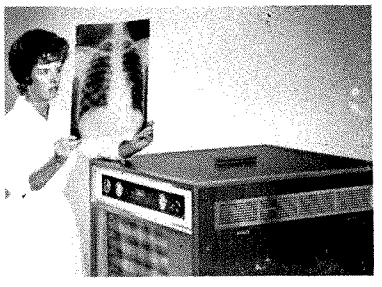
Materials. Many materials make up the parts and components of a KODAK X-OMAT Processor. Not one is there by chance; each one is there because it's the best one for its purpose that we know of.

Service. At the Kodak X-Omat Center there is a group of specialists whose sole concern is with KODAK X-OMAT Processors—every model, everywhere. KODAK X-OMAT Processor specialists check periodically with all X-OMAT Processor owners and dealers.

The Kodak X-Omat Center also administers a Process Control Program designed to assure each user periodically that his X-OMAT Processor is producing radiographs of top quality.

When an improvement is made, the word goes out to KODAK X-OMAT dealers and users—with full details on how to put it to use.

At the Kodak X-Omat Center there's a training school for dealers—how to install and service X-OMAT Processors. You can depend on these dealers for parts, and service "know-how." And, at the Kodak X-Omat Center, there's a school for technicians—how to operate and how to care for the equipment.



Except for the film-feeding station in the darkroom, the rest of the unit—about 35 inches in length—is in a lighted area.

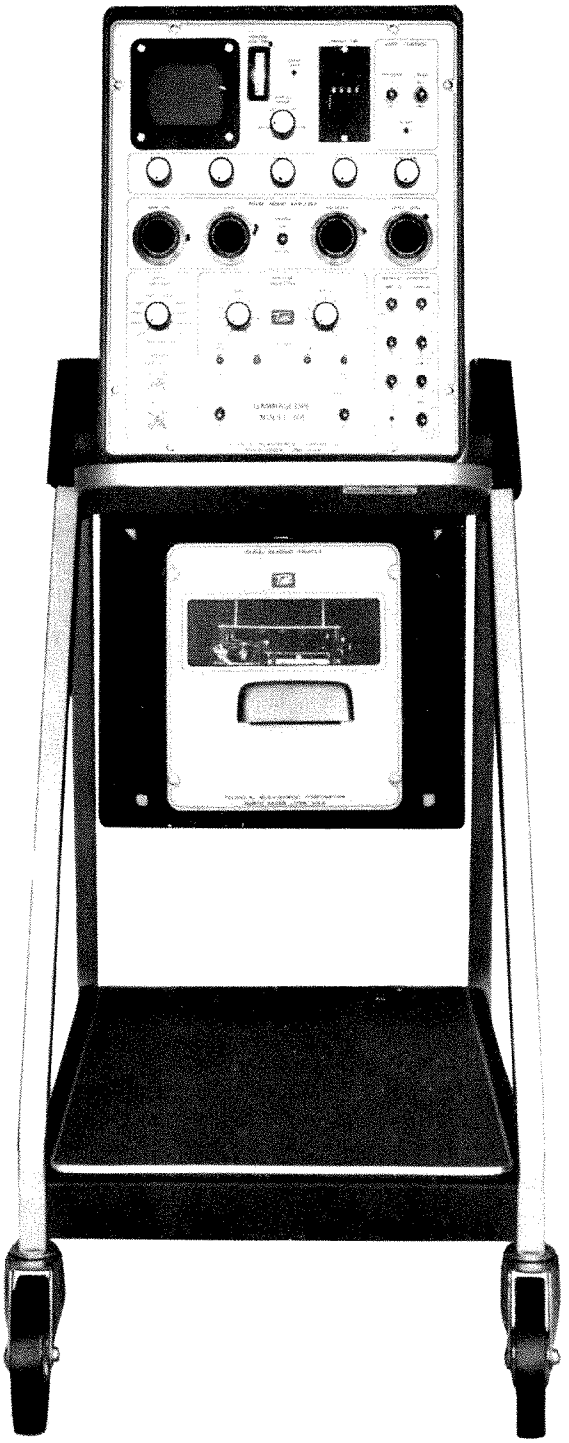
To get the best possible radiograph on the view box in the fastest time, you need the KODAK X-OMAT Processor, Model M4A. Furthermore, the installation of additional M4A units near "special procedures" rooms or gastrointestinal suites can increase over-all departmental efficiency.

To provide the same high-speed, dependable service for Emergency . . . Surgery . . . Neurology . . . Orthopedics, additional M4A units are the answer.

For information, consult your KODAK X-OMAT Processor dealer, or write

EASTMAN KODAK COMPANY
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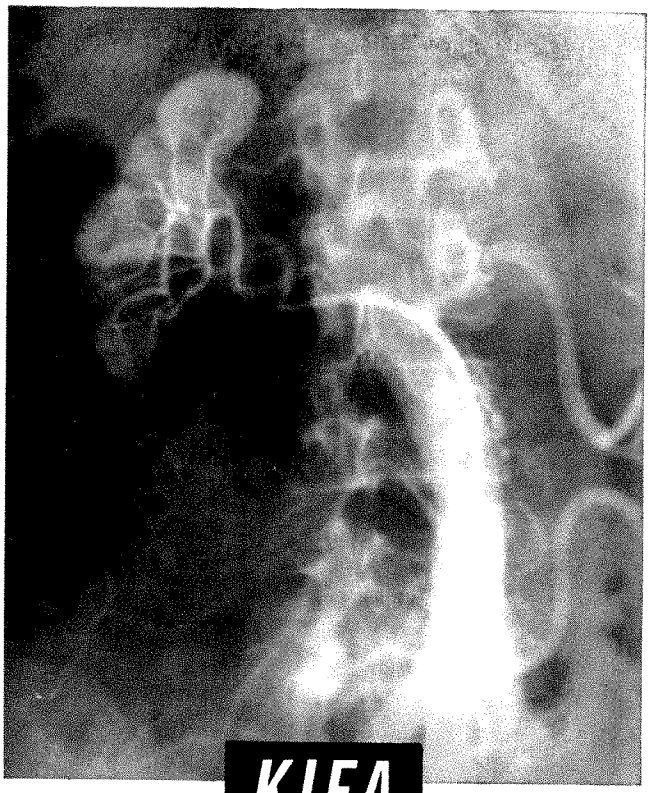


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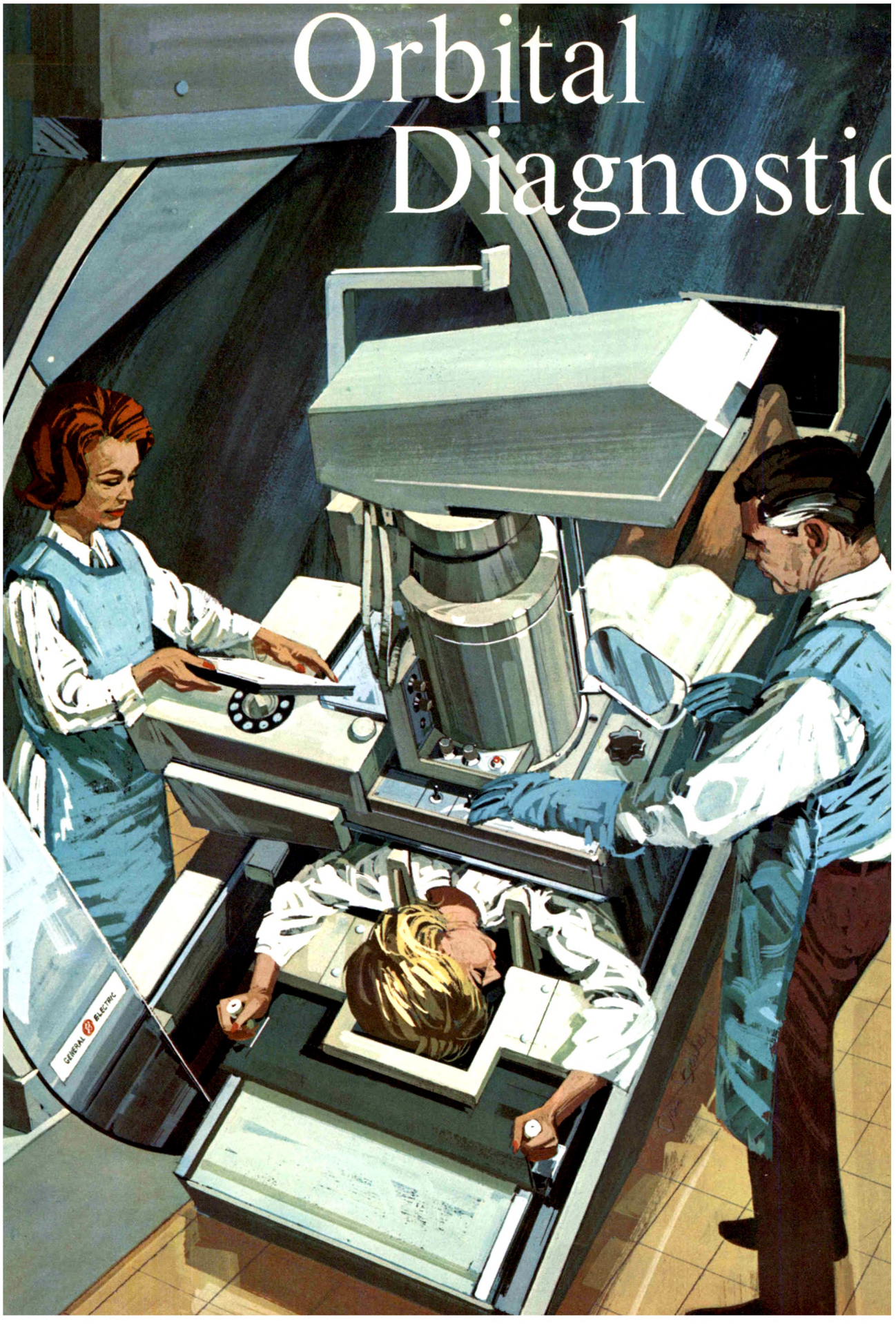
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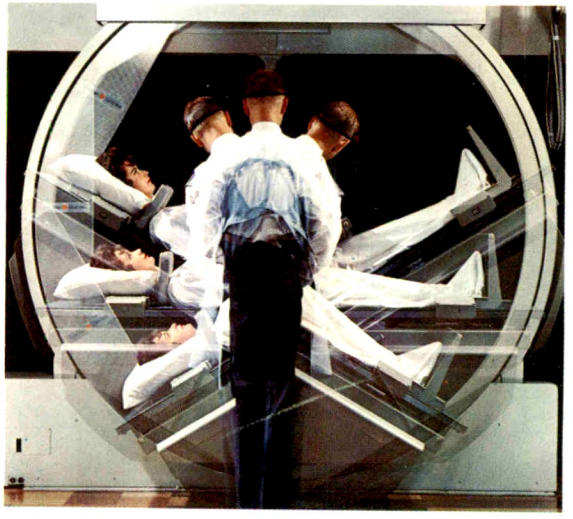
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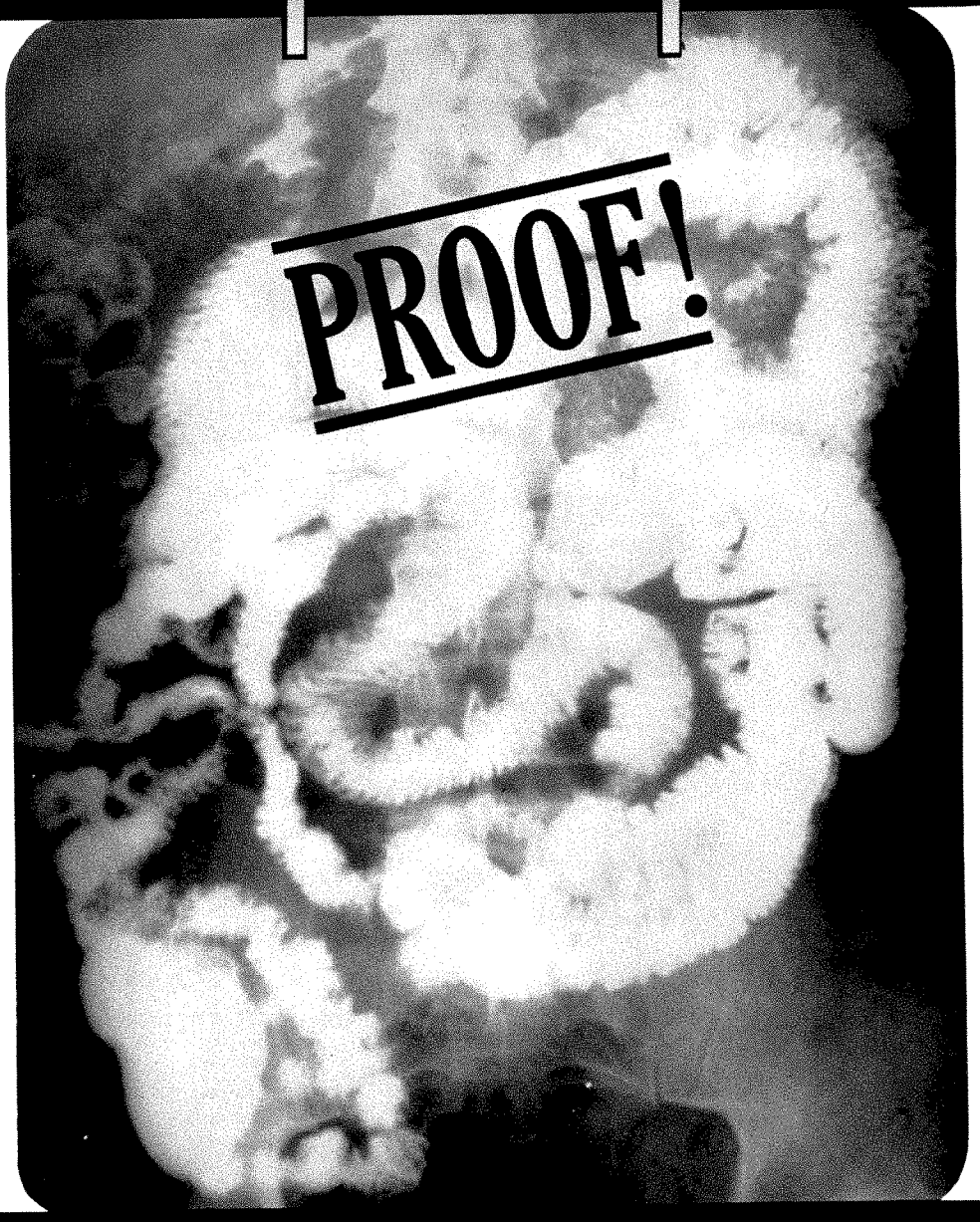
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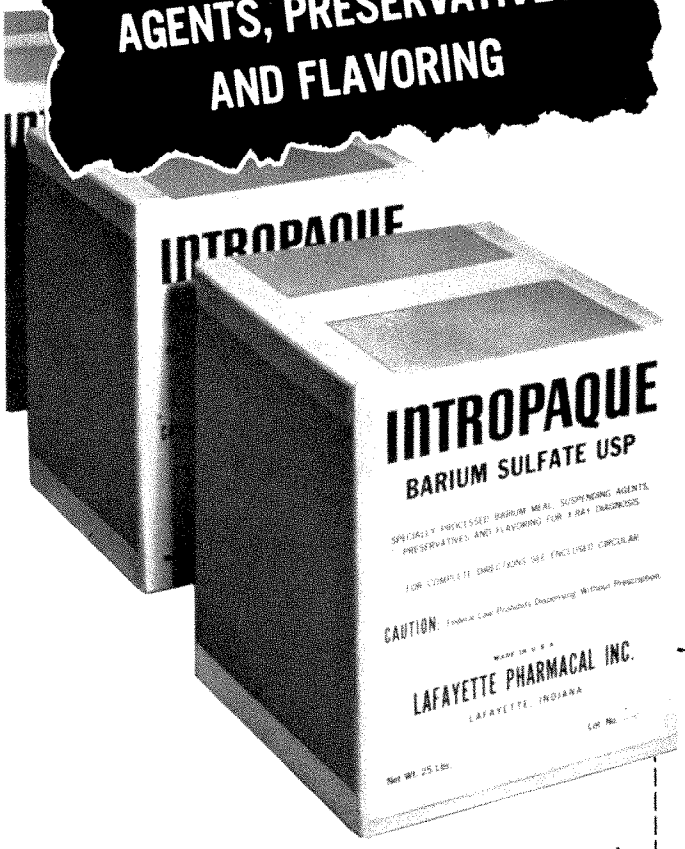
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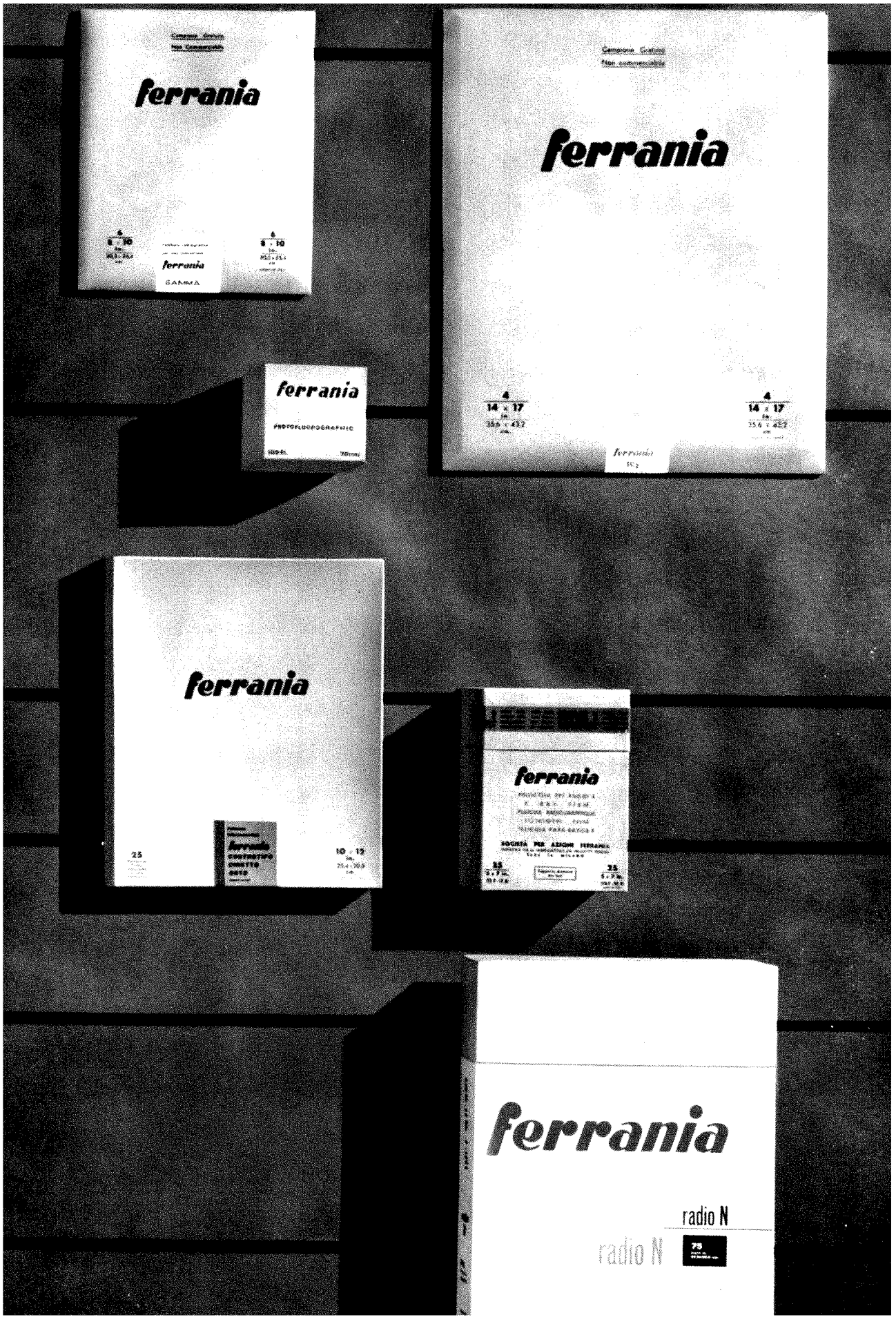
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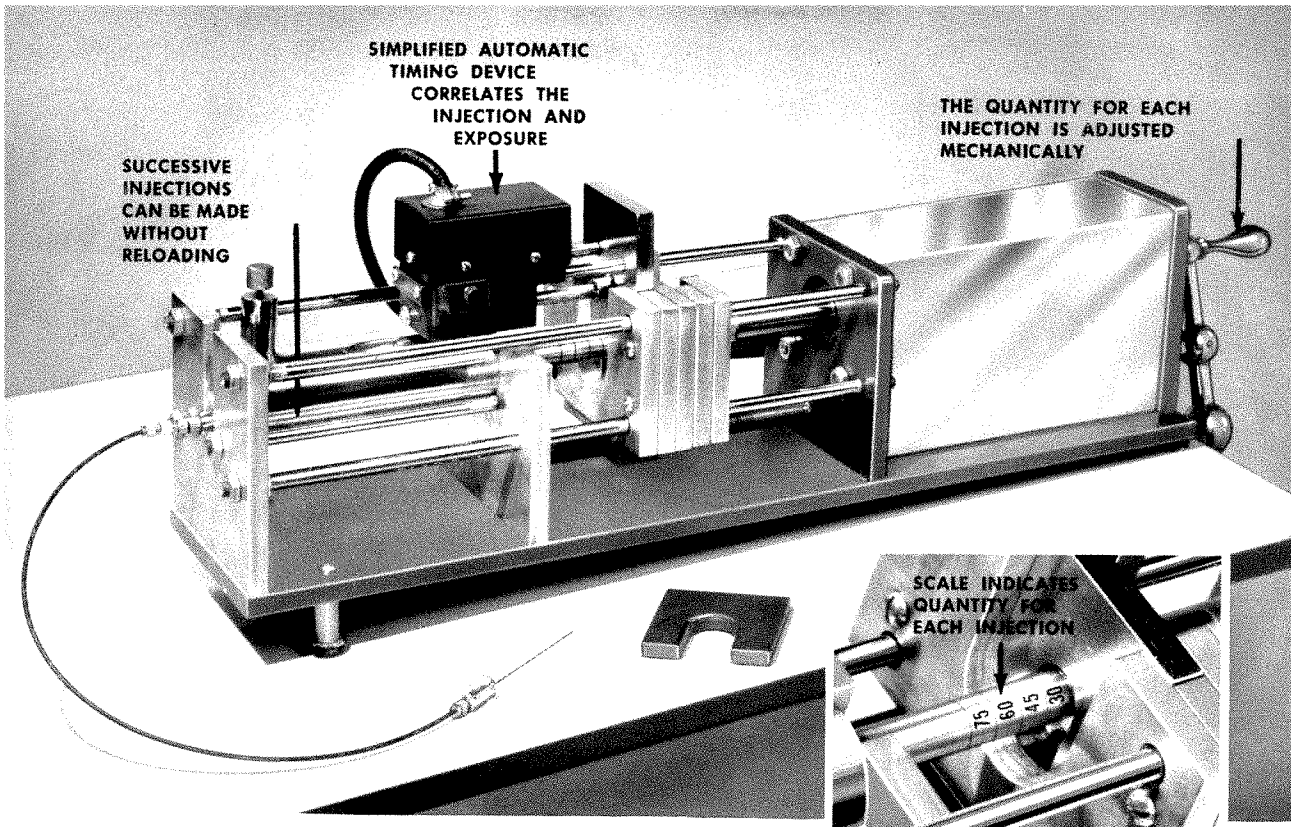
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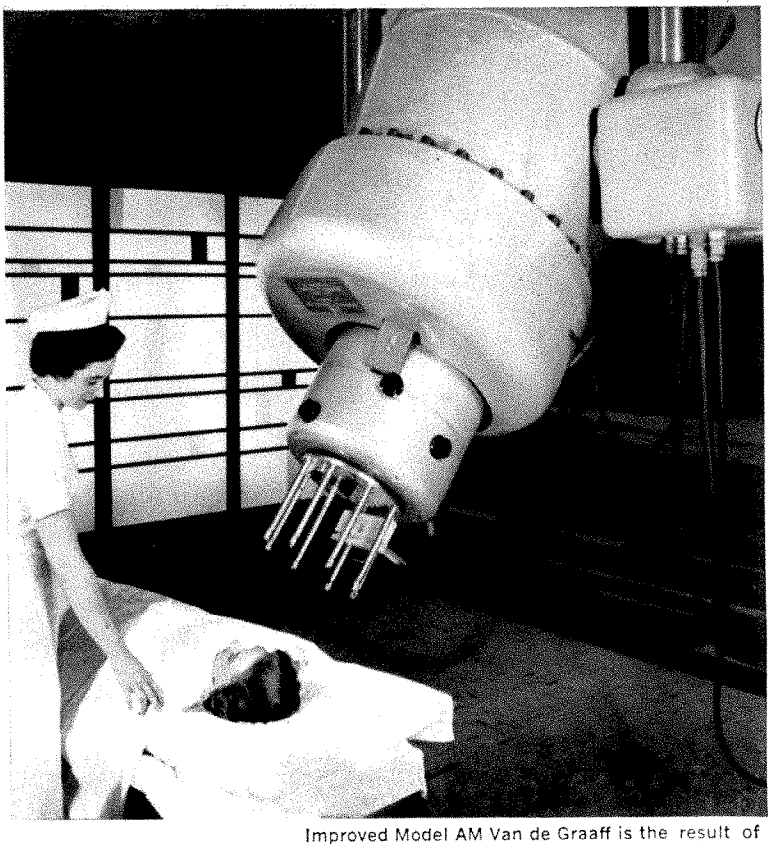
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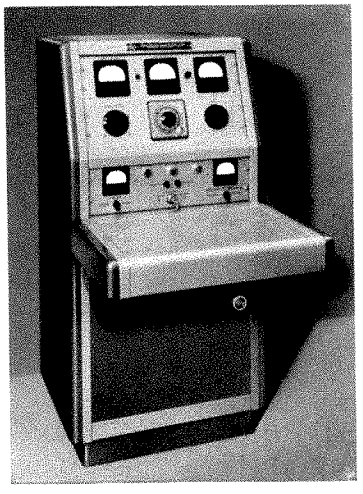


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THE AMERICAN JOURNAL OF ROENTGENOLOGY
RADIUM THERAPY AND NUCLEAR MEDICINE

VOL. 91 MAY, 1964 No. 5

ROENTGEN APPEARANCE OF THE SKELETON IN OSTEOMALACIA AND RICKETS*

By HOWARD L. STEINBACH, M.D., *and* MAX NOETZLI, M.D.
SAN FRANCISCO, CALIFORNIA

IN a previous study[21] it was noted that some patients with osteomalacia or rickets had dense instead of radiolucent skeletons. Other patients had roentgen evidence of secondary hyperparathyroidism, and because of the paucity of literature on the subject, it was thought that this was unusual. With increased experience in evaluating roentgenograms of patients with osteomalacia, it became apparent that these manifestations were more frequently present than at first assumed. They had, however, not always been identified. In order to determine the incidence of various roentgen signs that have been found in osteomalacia or rickets, roentgenograms of patients with this disease were re-examined.

Osteomalacia is a disorder of bone tissue in which calcium salts fail to deposit promptly in newly formed bone matrix. In growing children, in addition, impaired calcification of maturing cartilage at the zone of provisional calcification results in retardation of endochondral ossification. This condition is known as rickets. The retarded calcification of bone and cartilage matrix is not fully understood, but in some cases appears to be related to a diminished supply of vitamin D. Calcium deficiency produces rickets or osteomalacia only if vitamin D is also deficient. Other patients have an increased excretion of phosphate by the kidneys. A deficiency of alkaline phosphatase in the serum and tissues may impair the deposition of calcium phosphate salts and also result in osteomalacia or rickets. Some of the causes of osteomalacia and rickets are shown in Table 1.[1,14]

The malabsorption of vitamin D may be the result of (1) a deficiency of digestive enzymes such as in pancreatic disease; (2) a lack of emulsification of fat resulting from bile deficiency in liver disease; or (3) an inability of the small bowel mucosa to absorb fats as is present in sprue, neoplastic, inflammatory, or infiltrative disease of the small intestine or its mesentery. In these cases, absorption of calcium is frequently impaired. Calcium may combine with fatty acids to form compounds, not readily absorbed.

The cause for osteomalacia or rickets in

* From the Department of Radiology, University of California School of Medicine, San Francisco, California.
Presented at the Tenth International Congress of Radiology, Montreal, August 26–September 1, 1962.
This work was supported in part by Grant AM 02589 from the U. S. Public Health Service.

TABLE I

SOME CAUSES OF OSTEOMALACIA AND RICKETS

1. Vitamin D deficiency
2. Malabsorption from intestine
   - Small bowel
     - Sprue
     - Celiac disease
     - Pancreatic insufficiency
     - Chronic biliary obstruction or liver disease
     - Regional enteritis
     - Lymphosarcoma
     - Scleroderma
     - Amyloidosis
   - Postoperative complications
     - Small bowel resection
     - Enteric fistula
     - Blind pouch
     - Gastroenterostomy
     - Uterosigmoidostomy
   - Disease of mesentery
     - Carcinoma
     - Tuberculosis
     - Lymphoma
     - Amyloidosis
3. Excessive excretion of phosphorus or calcium by kidneys (congenital or acquired)
   - Renal tubular dysfunction with or without acidosis but without renal glomerular failure
   - Hyperthyroidism
   - Cushing's syndrome
   - Heavy metal poisoning
     - Lead
     - Beryllium
     - Magnesium, strontium or fluoride
4. Other causes
   - Hypophosphatasia (decreased alkaline phosphatase activity)
   - Hypoparathyroidism
   - Renal glomerular failure

patients with chronic renal glomerular failure is obscure. Some investigators believe that it results directly from acidosis, which inhibits the deposition of calcium salts in osteoid. The administration of alkali improves the bone lesions and alleviates bone pain.[4] A more likely explanation is that the uremic state antagonizes or interferes with the action of vitamin D. This is reflected in defective utilization of dietary calcium and phosphorus with a tendency toward lowering of the serum calcium level. The osteomalacia can be cured by the administration of large doses of vitamin D. This condition may thus be considered an acquired form of "vitamin D resistant rickets or osteomalacia."[19]

Renal disease affecting the tubules, without loss of glomerular function, is also frequently associated with rickets or osteomalacia. These lesions respond to very high doses of vitamin D and have been called vitamin D-resistant rickets. The kidney tubules normally reabsorb glucose, amino acids, sodium, calcium, phosphate and other substances. When this reabsorption is diminished for one or several of these substances, it produces phosphaturia, with or without amino aciduria or glycosuria. Renal phosphaturia produces a low plasma phosphorus which results in osteomalacia or rickets.

Biochemical evaluations more often reveal a normal than a low serum calcium and a low, rather than a normal, serum phosphorus. Usually the serum alkaline phosphatase is high. In hypophosphatasia, the alkaline phosphatase is low and the serum calcium and phosphorus may be normal or elevated. In patients with renal glomerular failure, the serum phosphorus is increased from diminished glomerular filtration rate. Calcium concentration in the serum is usually normal or low.

If osteomalacia is sufficiently far advanced, the usual roentgen appearance is that of generalized decreased bone density. When severe, the disease may be associated with bending or fractures of the weakened skeleton. Pseudofractures represented by radiolucent bands of diminished density of the bone cortex may be present, and are pathognomonic of osteomalacia. They are frequently multiple, in which case they may be almost symmetric. These pseudofractures have certain sites of predilection, such as the posterior and axillary borders of the scapulae (Fig. 1, *A* and *B*), the ribs, the pubic rami and ischia (Fig. 2), the medial aspect of the femoral neck (Fig. 3, *A* and *B*), the iliac bones, the radii, the ulnae, and the tibiae and fibulae (Fig. 4, *A* and *B*).

In growing children, additional changes occur at the sites of endochondral bone formation. The epiphyseal cartilage con-

tinues to proliferate and increase in thickness, but calcification of the mature, vacuolated cells and matrix does not keep pace. Consequently, the initial sign on roentgenograms is the presence of a wide space between the epiphysis and metaphysis with varying diminished density of the zone of provisional calcification. The width of the epiphyseal cartilage between the distal radial epiphysis and metaphysis in normal children is not usually greater than 1 mm. on roentgenograms. This was established by measuring the width in the center of the cartilage in 10 children of each year of age from 1 to 10 years. Similar measurements of 24 children with at least one sign of rickets of the distal radius or ulna ("cartilage widening," which in rickets is actually a combination of uncalcified cartilage and osteoid; diminished density or fraying of the zone of provisional calcification and cupping) ranged from 1.0 mm. to 10.0 mm. with an average of 3.2 mm. Only 6 of the 24 children had had spaces less than 1.5 mm. in thickness. Bone or calcified cartilage may continue to be formed at the outer margin of the metaphysis and extend around the periphery of the cartilaginous epiphyseal disk, producing on roentgenograms the appearance of cupping. Proliferation of cartilage and delayed calcification also take place around the margins of the epiphyses, but to a lesser extent. The rachitic changes are usually most obvious in those regions where bone grows rapidly or where it is subjected to pressure.

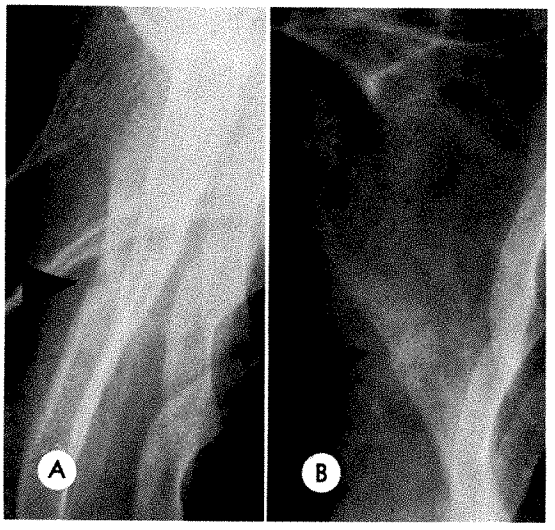


Fig. 1. (*A*) Tangential and (*B*) frontal views of the right scapula showing a pseudofracture along the posterior and axillary borders.



Fig. 2. Osteomalacia due to pancreatitis. Calcification is visible in the pancreas. Bilateral, almost symmetric pseudofractures of the superior and inferior rami of the pubic bones are present. The bones are diffusely demineralized.

The parathyroid glands regulate the calcium ion concentration in the blood. They respond directly to decreased calcium concentration in the serum by increasing in size and elaborating more parathyroid hormone, which, in turn, elevates the serum calcium. This elevation is accomplished in part by the direct action of the hormone on bone which, if sufficiently marked, produces osteitis fibrosa. These changes in bone may be recognized on roentgenograms when evidence of subperiosteal resorption of bone, large destructive bone cysts, or brown tumors, is present. The subperiosteal resorption of cortical bone may be generalized, but is frequently localized to certain sites of predilection. These are the thenar

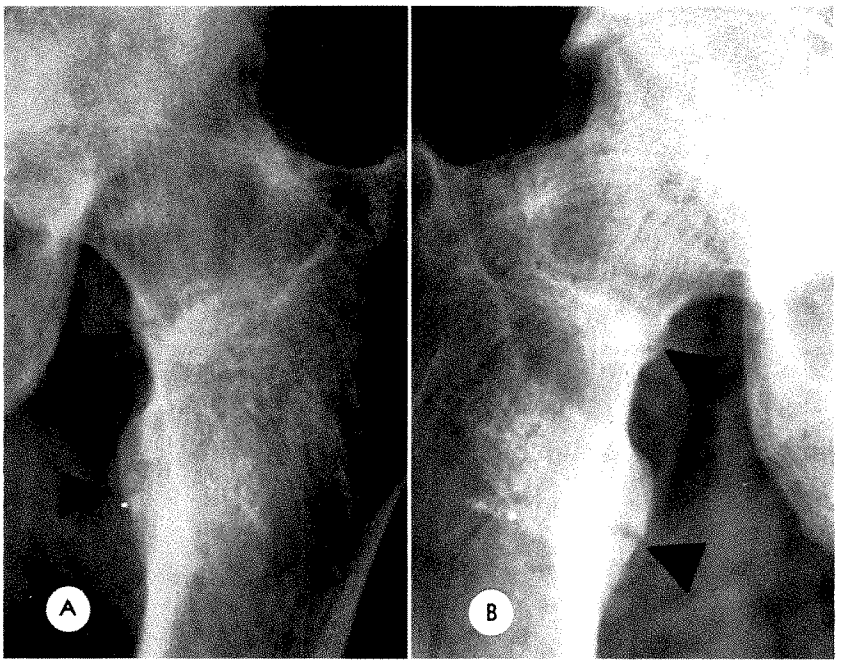


FIG. 3. (*A* and *B*) Bilateral, almost symmetric pseudofractures of the femoral necks, and pseudofractures involving the lesser trochanters.

surfaces of the middle phalanges of the fingers and the lamina dura of the jaws. Other sites are the outer surfaces of the clavicles, the upper medial surfaces of the tibiae, and the posterior and inferior surfaces of the calcanei. In addition, the sacroiliac joints, the necks of the humerus or femur, the ischial tuberosity, and the upper border of the ribs may be involved. These findings are regarded as pathognomonic of primary or secondary hyperparathyroidism. Hyperparathyroid bone lesions may occur whenever production of serum calcium is low.

In dietary or malabsorption osteomalacia, the situation exists for secondary hyperparathyroidism because of a low serum

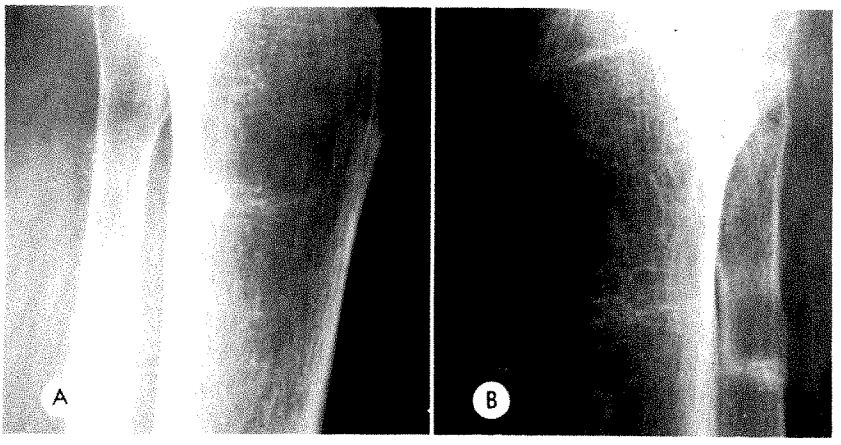


FIG. 4. (*A* and *B*) Bilateral, almost symmetric pseudofractures of the fibulae. The somewhat sclerotic margins, which are occasionally seen in osteomalacia, presumably are the result of an overgrowth of osteoid along the margins of the pseudofractures. Radiolucent lines in the center of the tibiae represent pseudofractures seen *en face*.

calcium which stimulates the parathyroids to secrete or raise the calcium to normal or near normal level. Since one of the actions of parathyroid hormone is to increase phosphate excretion by inhibiting resorption of phosphate by the kidney tubules, the serum phosphate is low.

The serum calcium level is also low in renal glomerular failure, thus producing osteitis fibrosa through stimulation of the parathyroids. In these cases the serum phosphorus level is elevated because of the reduced glomerular filtration rate.

METHOD

The presence of osteomalacia was established on clinical data in all cases and in many was confirmed by bone biopsy. Roentgenograms of 25 adult patients with osteomalacia and of 27 children with rickets were reviewed. The disease in 17 of the adult patients and in 2 of the children was attributed to malabsorption. Malabsorption in the 17 adults resulted from sprue (12); pancreatic insufficiency (2); gastroenterostomy (1); regional enteritis (1); and in 1 the cause was unknown. Only 2 children with vitamin D deficiency had sufficiently thorough roentgen examinations to be included.

Seven adults had osteomalacia and 24 children had rickets as a result of renal abnormalities. All of the adults and 13 of the children had renal tubular insufficiency of varying severity. Eleven children had renal glomerular failure. Using the classification of Fraser and Salter[15] modified to include adults (Table II), these cases may be classified as follows. All 7 adults had simple hypophosphatemic vitamin D-refractory osteomalacia (Type III, A, i). Ten children had simple hypophosphatemic vitamin D-refractory rickets (Type III, A, i); 1 with amino-aciduria (Type III, A, ii); 1 with amino-aciduria and acidosis (Type III, A, iii); and 1 with cystine storage disease (Type III, A, iv).

Impaired glomerular excretion causing renal disease resulted in renal osteodystrophy in 11 children (Type II).

RESULTS

I. ADULTS

A. Osteomalacia Due to Malabsorption of Vitamin D (*Table* III). All except 1 of the 17 patients had diminished skeletal density. Nine had typical pseudofractures which were regarded as diagnostic of the disease. Five had roentgen evidence of hyperparathyroidism, manifested by subperiosteal resorption of cortical bone. This resorption was present in the middle phalanges in all 5 and involved the tufts of the distal phalanges or other cortical sites as well (Fig. 5). In addition, 3 patients had cyst-like bone destruction at various sites (Fig. 6). Two of these 5 patients had no pseudofractures, presenting a roentgen appearance of hyperparathyroidism alone. One patient with diminished skeletal density, pseudofractures, subperiosteal resorption, and localized areas of bone destruction had steatorrhea and a parathyroid adenoma. A similar case was reported by Davies, Dent, and Willcox.[8]

All 17 patients were women. The patients without hyperparathyroid bone changes ranged in age from 26 to 72 years with half of them being older than 60. Those with secondary hyperparathyroidism tended to be younger, with ages ranging from 30 to 72 years; three-fourths of them were less than 40.

B. Osteomalacia Due to Renal Tubular Insufficiency Resulting in Phosphate Depletion (*Vitamin D-Resistant Osteomalacia*) (*Table* IV). All 7 of the adult patients without malabsorption defects or hypophosphatasia were in this category. Three of these patients had diminished skeletal density, whereas 4 had increased thickness and density of the bones (Fig. 7 and 8, *A* and *B*). The bones of all 7 were short, indicating that the disease had been present in childhood. Pseudofractures were visible in 3 patients. None had roentgen signs of hyperparathyroidism. Extensive localized overgrowth of bone in the form of accessory ossicles, spurs, and exostotic growths, which has been found only in this form of osteo-

malacia, was present in 5 patients. The small ossicles or extra centers of ossification were present in many of the joints but in all cases involved the wrists just distal to the radius and ulna. They were frequently seen in the anterior aspect of the elbow joint and about the hips (Fig. 9). The adjacent cartilage was frequently narrow. The exostotic growths were in the regions of ligamentous or tendinous insertions and were seen in periarticular areas.

C. Osteomalacia Due to Hypophosphatasia. One adult with hypophosphatasia had diminished density of her skeleton and

TABLE II

CLASSIFICATION OF RICKETS AND OSTEOMALACIA
(Modified from Fraser and Salter[15])

Type	Etiology	Designation Preferred by Authors	Other Designations for the Syndrome	No. of Cases (present series)		No. of Cases (Fraser and Salter, 1946–1955[15])
				Child.	Adults	
		Responsive to usual therapeutic doses of vitamin D				
I. *Vitamin D deficiency*						
i.	Dietary deficiency of vitamin D	Vitamin D deficiency rickets or osteomalacia	Osteomalacia and rickets, classical rickets or osteomalacia, infantile rickets, simple rickets or osteomalacia, deficiency rickets	1	0	70
ii.	Defective absorption of vitamin D and calcium due to steatorrhea	Malabsorption rickets or osteomalacia	Celiac rickets, malabsorption rickets or osteomalacia	2	17	
II. *Chronic renal insufficiency with secondary hyperparathyroidism*	(Chronic pyelonephritis, chronic glomerulonephritis, congenital renal abnormalities, late stages of Type III, A, iii, iv; B, i)	Renal osteodystrophy	Renal rickets, renal osteodystrophy, rickets due to glomerular insufficiency, glomerular rickets	11		6
		"Resistant" to usual doses of vitamin D				
III. *Renal tubular insufficiency*						
A.	Renal tubular insufficiency resulting in phosphate depletion					
i.	Defective resorption of phosphate (± glucose)	Hypophosphatemic vitamin D-refractory rickets or osteomalacia of the simple type	Vitamin D-resistant rickets or osteomalacia (with hypophosphatemia), primary vitamin D-refractory rickets or osteomalacia, tubular rickets or osteomalacia, endogenous rickets or osteomalacia, late rickets, rachitis tarda, phosphate diabetes, Milkman's syndrome (adult), idiopathic osteomalacia (adult)	7	10	14
ii.	Defective resorption of phosphate, glucose, many amino-acids (± protein)	Hypophosphatemic vitamin D-refractory rickets or osteomalacia with amino-aciduria	Fanconi syndrome, Debré-deToni-Fanconi syndrome, nephrotic glycosuric dwarfism with hypophosphatemic rickets, amine-diabetes	1	2	
iii.	Defective resorption of phosphate, glucose, many amino-acids, protein (± water), with defective conservation of fixed base	Hypophosphatemic vitamin D-refractory rickets or osteomalacia with amino-aciduria and acidosis		1		0
iv.	Generalized defect in cystine metabolism, with (? secondary) defective tubular resorption of phosphate, glucose, many amino-acids, protein, fixed base (± water)	Cystine storage disease with vitamin D-refractory rickets	Fanconi syndrome, Debré-deToni-Fanconi syndrome, Lignac's disease, Lignac-Fanconi syndrome, cystinosis, cystinuria with rickets	1		4
v.	Defective resorption of phosphate, glycine and fixed base	Hypophosphatemic vitamin D-refractory rickets with hyperglycinuria				0
vi.	Miscellaneous abnormalities with associated renal tubular resorption defects	Oculo-cerebro-renal syndrome with hypophosphatemic vitamin D-refractory rickets, other rare conditions	Organic aciduria, decreased renal ammonia production, hydrophthalmos and mental retardation			0
B.	Defective handling of other ions by the renal tubules resulting in calcium depletion					
i.	Defective conservation of fixed base	Rickets or osteomalacia due to renal tubular acidosis	Infantile renal acidosis, Lightwood's syndrome, hyperchloremic renal acidosis (with nephrocalcinosis), Albright's syndrome (adult)	0		0
ii.	Defective calcium resorption		Rickets or osteomalacia due to idiopathic hypercalcuria			0
IV. *Miscellaneous*						
i.	Defective calcification of bone associated with generalized depression of alkaline phosphatase activity	Hypophosphatasia	Osteodysmetamorphosis fetalis, osteoblastic dysostosis, rickets with low alkaline phosphatase activity	1		4

multiple pseudofractures, as well as true fractures. Because the bones were not deformed and were of normal length, it is assumed that the disease was acquired after growth had ceased.

II. CHILDREN

Roentgenograms of 27 patients were evaluated and the results tabulated (Table v). Besides evaluating density, pseudofractures, subperiosteal resorption, accessory ossicles, and exostotic growth, we were concerned with the various abnormalities occurring only in children. These abnormalities were widened epiphyseal cartilages, fraying and cupping of the metaphyses, bending of the bones, subperiosteal new bone formation, and retarded maturation of the skeleton.

A. Rickets Due to Vitamin D Deficiency (*Table* v, *Group* 1). Only 1 patient with dietary vitamin D deficiency (Group 1, i) and 2 with malabsorption due to biliary atresia (Group 1, ii) had reasonably complete skeletal surveys. These 3 patients showed the usual demineralization, thickened epiphyseal cartilages, fraying and cupping of metaphyses, bending of bones,

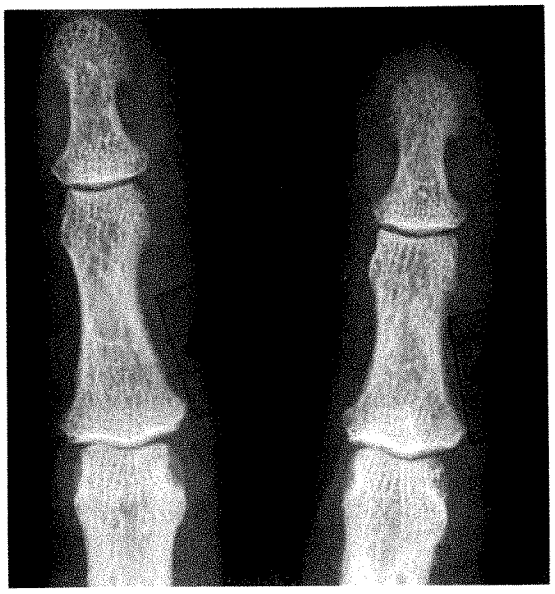


Fig. 5. Detailed view of the second and third fingers of the left hand of a patient with osteomalacia due to regional enteritis. The arrows point to areas of subperiosteal resorption involving the cortex of the middle phalanges. The changes are more severe on the surface facing the thumb. Minimal resorption of the terminal tuft of the distal phalanx of the second finger is seen.



Fig. 6. Destructive cyst-like lesion of the anterior aspect of the tibia due to osteitis fibrosa in a patient with osteomalacia. This lesion healed after the patient received large doses of vitamin D.

Table III

ROENTGEN FINDINGS IN MALABSORPTION OSTEOMALACIA

(17 Patients—All Females)

Demineralization			16
Demineralization alone	6		
Demineralization and pseudofractures	6		
Demineralization, pseudofractures, and hyperparathyroidism*	3	5 patients with secondary hyperparathyroidism	
Demineralization and hyperparathyroidism	1		
Normal density			1
Hyperparathyroidism only	1		

* One of these patients also had a parathyroid adenoma.

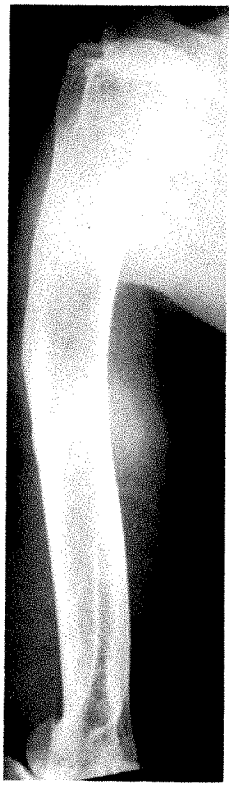


FIG. 7. Roentgenogram of the right humerus in a patient with osteomalacia from renal tubular insufficiency which resulted in phosphate depletion (vitamin D-resistant osteomalacia). The humerus is broad and short and shows an increased bowing in its upper part. The bones are of increased density. The cortex of the shaft has lost its compact character and the trabeculae are increased in thickness.

and delayed skeletal maturation typical of rickets. One patient with biliary atresia had pseudofractures and one with dietary deficiency had periosteal new bone formation. Both patients with biliary atresia had visible clubbing of the fingers. None had roentgenographic evidence of secondary hyperparathyroidism.

B. Rickets Due to Chronic Renal Insufficiency (*Table* V, *Group* II). There were 11 patients with severe chronic renal failure and abnormal bones (Group II). Diminished density of part of the skeleton was present in 10 patients. The density of the bones varied in different parts of the skeleton with a relatively increased density, usually in the spongy bone of the metaphyses, present in 9 patients (Fig. 10). This associated increased and decreased density was frequently well seen in the vertebral bodies. Broad dense bands in the upper and lower portions of the bodies, with radiolucent centers, are seen only in this form of osteomalacia or rickets. Pseudofractures were present in only 1 patient.

Subperiosteal resorption, always involving the middle phalanges of the fingers, but frequently involving other bones, was identified in 9 patients and was questionable in the other 2 (Fig. 11). In the roentgenograms of these 2, intensifying screens were used and the bone detail was not as well seen as in those without screens. Roentgenograms of the hands of 1 patient showed no subperiosteal resorption when screens were used, but unquestionable resorption without the screens. One patient had a localized, cyst-like area of destruction in the distal radius. The skeleton in patients with chronic renal failure is more likely to demonstrate characteristic changes of hyperparathyroidism than other forms of osteomalacia or primary hyperparathyroidism.[9]

Widening of the epiphyseal cartilage was the most common manifestation of rickets and was seen in 7 of the 9 patients with unfused epiphyses. Rachitic rosary of the chondral border of the ribs was visible in 7, and fraying or cupping, or both, was seen in the metaphyses of 5. Maturation and growth were delayed in 8 patients, which is to be expected in any chronic disabling disease. In 1 patient, growth was severely retarded but the maturation of the skeleton was normal. This dissociation of matura-

TABLE IV

ROENTGEN FINDINGS IN ADULTS WITH RENAL TUBULAR INSUFFICIENCY WITH OSTEOMALACIA
(5 Males, 2 Females)

A. Diminished density		3
B. Increased density		4
C. Pseudofractures		3
With decreased density	1	
With increased density	2	
D. Accessory ossicles, spurs, and exostoses		5

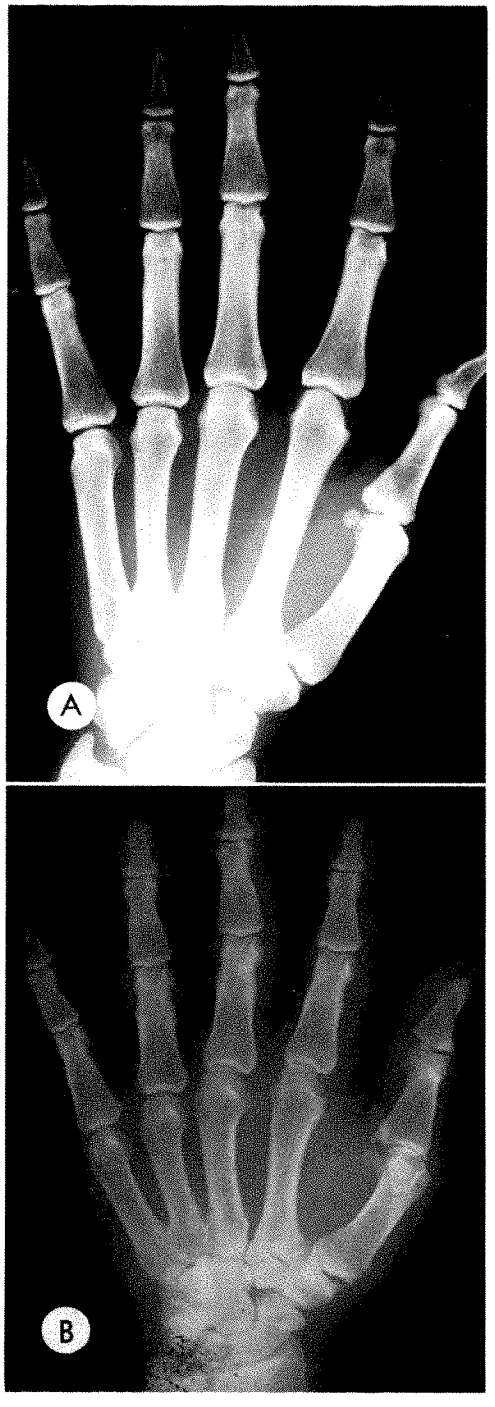


Fig. 8. (*A* and *B*) Roentgenograms of hands of 2 patients with osteomalacia due to renal tubular insufficiency which resulted in phosphate depletion (vitamin D-resistant osteomalacia). Both patients have dense bones. Multiple exostoses are present primarily at the ends of the tubular bones in the region of the insertion of tendons and ligaments. Small extra centers of ossification, characteristic of this form of osteomalacia, can be seen in the wrist joints between the radius and ulna and between the carpal bones.

tion and growth is not uncommon in chronic renal failure. Extensive calcification of the soft tissues, particularly of periarticular structures and vessels, was present in only 1 patient (Fig. 11 and 12). Another patient had slight vascular calcification. One patient had bilateral slipping of the femoral and humeral heads (Fig. 13).

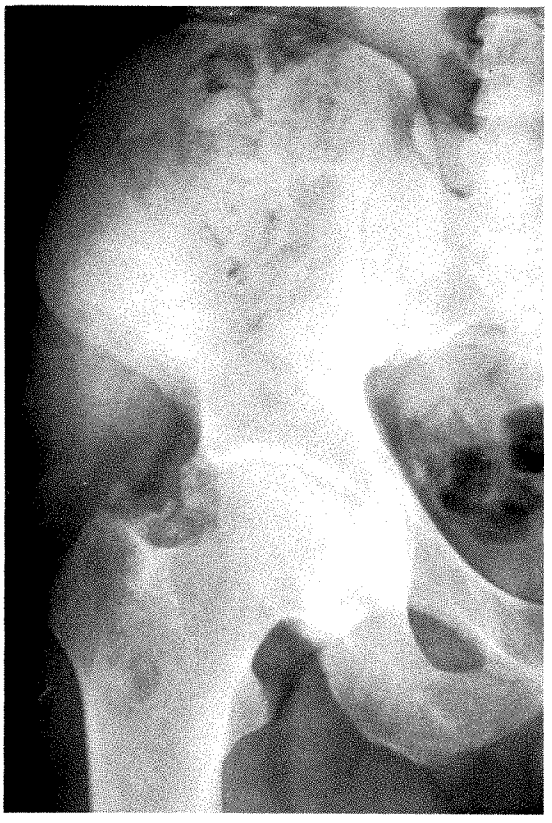


Fig. 9. Renal tubular insufficiency in this patient resulted in phosphate depletion (vitamin D-resistant osteomalacia). Exostoses and separate centers of ossification are present along the lateral and medial aspects of the hip joint.

C. Rickets Due to Renal Tubular Insufficiency (*Table* v, *Group* iii). Thirteen patients with renal tubular insufficiency of varying severity were studied. The most



FIG. 10. Roentgenogram of the femur and tibia of a child with chronic renal insufficiency due to pyelonephritis. The over-all density of the bones is increased but areas of radiolucency are scattered throughout the spongiosa. Subperiosteal resorption of the medial aspect of the upper end of the tibia indicates secondary hyperparathyroidism. No roentgenographic evidence of rickets is seen but the epiphyses are almost fused.

common (10 cases) was the simple type (III, A, i) with phosphate depletion, with or without glucosuria. This disease is also known as vitamin D-resistant rickets, late rickets, and phosphate diabetes. The roentgen appearance in some cases differed from other types of rickets in that the bones were of increased density. Three of the patients had thick dense bone on initial examinations. In 3 others it developed after prolonged observation and after varying doses of vitamin D, although signs of rickets were not completely alleviated. The other 5 patients whose disease was of a more severe form had diffuse demineralization. The most common sign of rickets was increased thickness of the epiphyseal cartilages, most prominent about the knees. Since rickets is a disease of growing bones, it would be expected initially at the distal femoral metaphysis which is the fastest growing area of the skeleton. The proximal and distal tibial metaphyses are next in rate of endochondral ossification, with the distal radius and



FIG. 11. Roentgenogram of the hand of a child with chronic renal insufficiency resulting from pyelonephritis. The metacarpals are disproportionately short in comparison with other tubular bones. The patient has evidence of secondary hyperparathyroidism with resorption of the cortex of the medial phalanges, which is not clearly visible in this reproduction. Some resorption of the tufts of the distal phalanges, particularly of the first and third fingers, is present. Soft tissue in the periarticular regions and vessels is calcified. No definite evidence of rickets is seen in the radius and ulna, although the epiphyseal cartilages of the first metacarpal and of all of the proximal phalanges appear slightly widened. The bones are dense with areas of rarefaction scattered throughout.

TABLE V

EVALUATION OF ROENTGENOGRAMS OF 27 CHILDREN WITH RICKETS

Group (Fraser and Salter Classifications[35])	No. of Patients	Demineralization	Dense Bones	Coarse Texture	Pseudo-Fracture	Subperiosteal Resorption	Periosteal New Bone	Bowing	Widened Epiphyseal Cartilage	Fraying and Cupping	Rosary	Bone Age
I. (i)	1	1	—	—	—	—	Marked	1	1	1	1	Not evaluated
(ii)	2	2	—	1	1	Roentgenograms inadequate	1	1	2	2	1	2 delayed
II.	11	10	9	10	1	0	0	3	7	5	7	8 delayed
III. A. (i)	10	5	6	5	—	—	3	8	9	7 1 Partial	7 1 No roentgenogram	3 delayed
(ii)	1	1	—	—	—	—	—	—	1	1	1	Delayed
(iii)	1	—	—	—	—	—	—	1	—	—	No roentgenogram	Marked delay
(iv)	1	1	1	1	—	1	—	—	1	1	—	Marked delay

ulna and proximal humerus following. In 1 patient, the distal femur and proximal tibia showed definite disease, but the distal radii and ulnae appeared normal (Fig. 14, *A* and *D*). The other patients had more severe disease with most of the bones involved.

Another factor influencing the cartilage thickness and rachitic changes is stress. In the normal child the femoral epiphyseal cartilage on the medial aspect is thicker than the lateral because of more stress during weight-bearing. In mild rickets, the initial stages of the disease may be obvious only in the medial metaphyseal area of the femur (Fig. 15, *A* and *B*). Caffey[6] stated that infants who crawl and thus bear more weight on their upper extremities may have greater rachitic changes in the distal radius or ulna than in the distal femur.

In 7 patients, the metaphyses of the long bones together with the costochondral junction were frayed and cupped. Bones of increased density and thickness, as well as those of diminished density, showed bending of varying degree in 8 patients. Of particular interest in this group was excessive periosteal bone formation, particularly at the sites of muscle, ligamentous, or tendinous insertions. This increased with the age of the child. In some instances this proliferation produced exostotic growth of the type seen in the adult patients with osteomalacia. The extra ossification centers about the radiocarpal articulations and other joints were not seen in any of the children. In the wrist of 1 patient, however, extra centers developed after the epiphyses had closed. The first manifestation was a small exostotic growth arising from the ulnar side of the radial epiphysis, which then became detached. Pseudofractures did not occur in

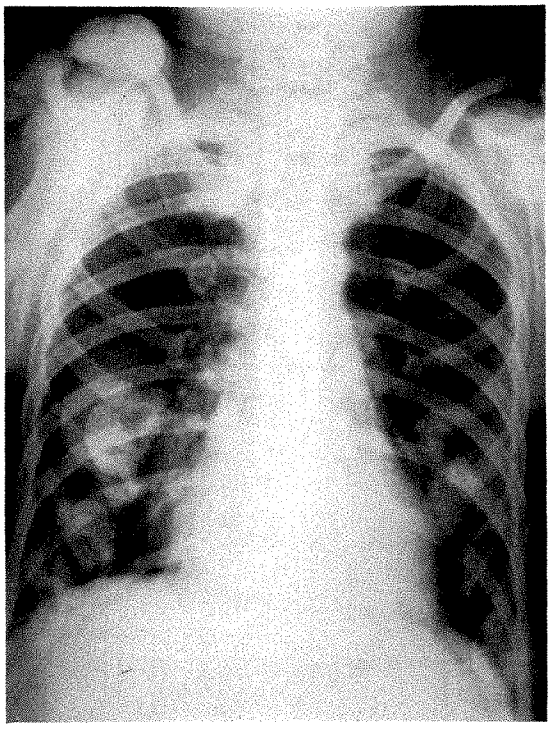


FIG. 12. Same patient as in Figure 11. Roentgenogram shows extensive calcification about the acromioclavicular joints and at the ends of the ribs. Slipping of the heads of both humeri is present.

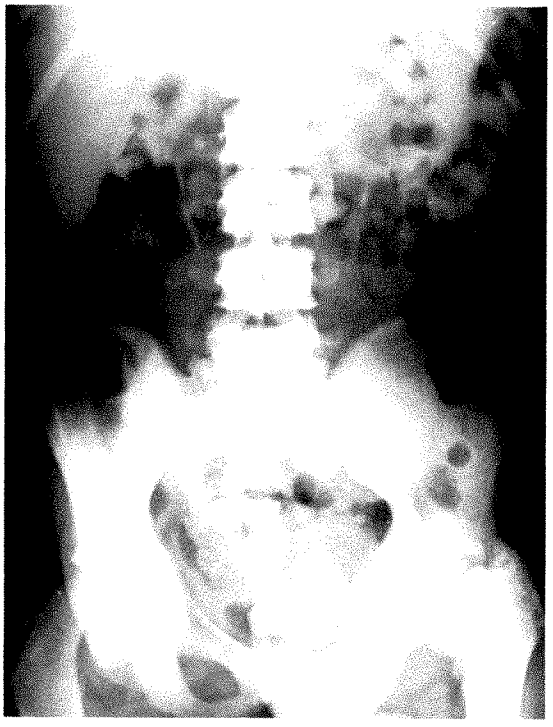


FIG. 13. Same patient as in Figure 11. Bilateral slipped femoral epiphyses.

any of the 10 patients. The bone age was delayed in 3.

Two patients with more severe renal tubular dysfunction were studied. One had amino-aciduria (Type III, A, ii) and the other, amino-aciduria and acidosis in addition to hyperphosphaturia and glucosuria (Type III, A, iii). These patients had the typical rachitic changes of demineralization, widened epiphyseal cartilage, and metaphyseal fraying and cupping. Pseudofractures, subperiosteal resorption, or excessive periosteal bone formation were absent. Bone maturation was markedly retarded. The patient with acidosis also had nephrocalcinosis, which is common in this disease.

One patient with severe, generalized tubular resorptive defect and cystine storage disease (Fanconi syndrome, Debré-deToni-Fanconi syndrome, Type III, A, iv) had severe renal glomerular damage in addition to the renal tubular defect (Fig. 16, *A* and *B*). The bone abnormalities were those seen

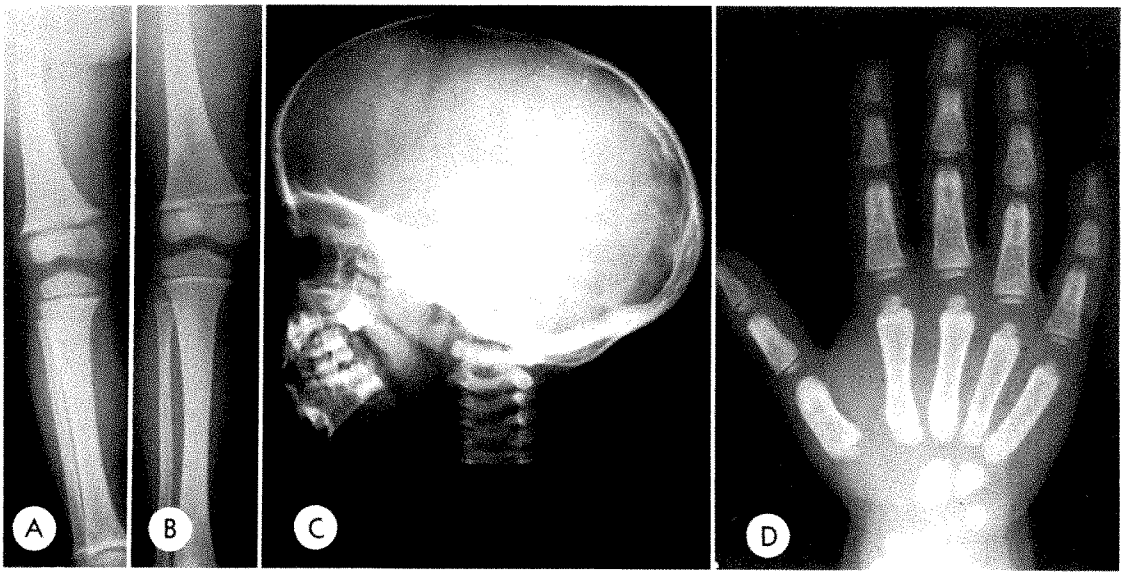


FIG. 14. Rickets due to renal tubular insufficiency resulting in phosphate depletion (vitamin D-resistant rickets). (*A*) Roentgenogram of the right lower extremity before the patient was treated with large doses of vitamin D. Slight widening of distal femoral epiphyseal cartilage, which can be seen to a lesser extent in the epiphyseal cartilages of the proximal and distal ends of the tibia. (*B*) With treatment, the epiphyseal cartilage reverted to normal thickness and areas of increased density are seen in the region with zones of provisional calcification. (*C*) The elongation of the skull is produced by premature synostosis of the sagittal suture. (*D*) In the roentgenogram of the wrist, the cartilage of the epiphyseal plate appears of normal width, which is in contrast to that of the femoral and tibial epiphyseal cartilage, mentioned in *A*.

with chronic renal glomerular damage. Evidence of rickets in the metaphyseal area of the distal end of the femur in association with subperiosteal cortical resorption of the phalanges indicated secondary hyperparathyroidism. In primary hyperparathyroidism, subperiosteal resorption is present but there is no evidence of rickets or osteomalacia. The bones varied in density as seen in patients with chronic renal glomerular damage.

DISCUSSION

From this study it is apparent that osteomalacia or rickets presents 3 distinct roentgen patterns. These patterns are dependent upon the basic metabolic defect responsible for the bone disease.

I. Malabsorption and Dietary Deficiencies. This group usually shows the classic decreased density of the skeleton which involves both the peripheral and the axial skeleton, as well as the cortex and the spongiosa. About half of the patients in this group had pseudofractures which, when typical in location, were considered pathognomonic of the disease. Surprisingly, 5 of the 17 adults had roentgen signs of hyperparathyroidism (Fig. 1–6 inclusive). Previous reports of secondary hyperparathyroidism associated with malabsorption osteomalacia have appeared in the literature,[8,21,22] but it has been considered an unusual finding. To detect early hyperparathyroidism, it is necessary that roentgenograms of the hands be obtained, preferably without intensifying screens. Resorption is usually initially present along the radial surface at the base of the shaft of the middle phalanges. In some cases, all of the middle phalanges are involved, whereas in others, only one finger of one hand may show resorption. The compact structure of the involved cortex frequently develops vertical striations and the outer surface becomes roughened.[20] Subperiosteal resorption may be seen in other sites, such as the lamina dura of the jaws, distal ends of the radius or ulna, proximal medial tibia, ends of the clavicle, neck of the femur, posterior and inferior margins of the calcaneus, and in the upper border of the ribs. In severe cases, subperiosteal resorption may involve the cortex of most of the bones. Two of the patients with hyperparathyroid bone changes did not have pseudofractures. Primary hyperparathyroidism might have been suspected had the clinical and laboratory data not been available.

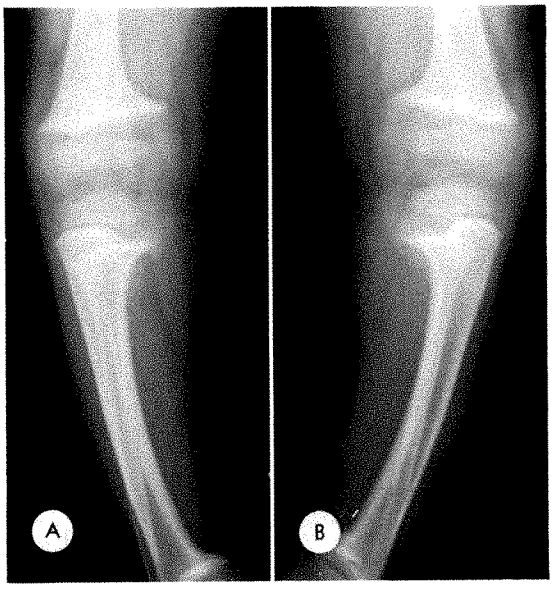


Fig. 15. (*A* and *B*) Osteomalacia due to renal tubular insufficiency resulting in phosphate depletion (vitamin D-resistant osteomalacia). Widening of the epiphyseal cartilages of the distal end of the femora and of the proximal and distal ends of the tibiae is seen almost exclusively along the medial aspects. The bones are dense.

Two children with biliary atresia had clubbing of the fingers in addition to the usual signs of rickets. Both clubbing and rickets, or osteomalacia, are seen in severe liver disease.

II. Renal Tubular Insufficiency (Vitamin D-Resistant Osteomalacia or Rickets). The simple, mild type with the reabsorption defect limited to phosphate and sometimes to glucose is by far the more common. It was present in all of the adults and in 10 of the 13 children. In our experience, slightly more than half of the adults and children had bones of increased, rather than of decreased, density. This was never seen in the malabsorption or dietary cases. The diame-

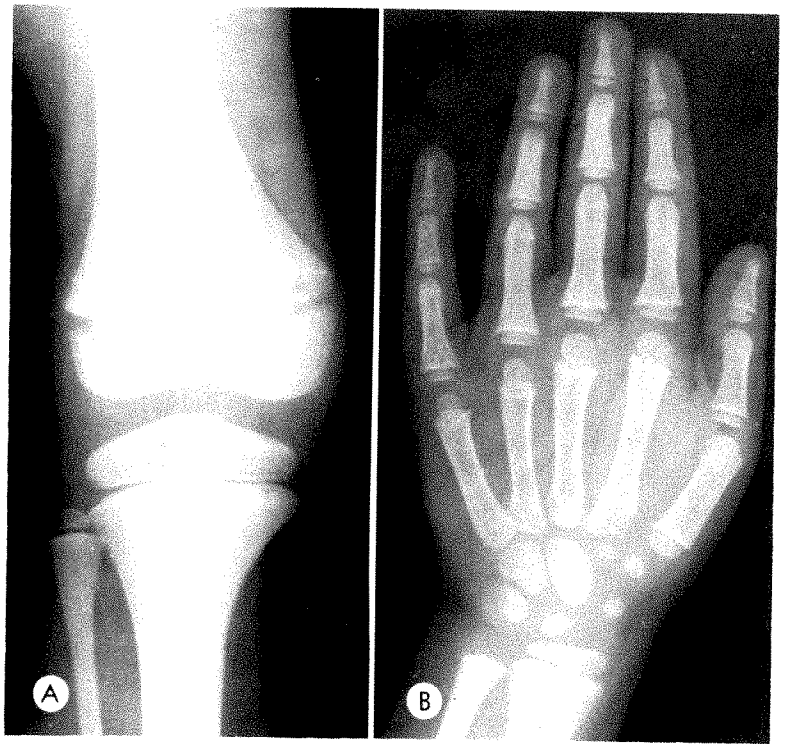


Fig. 16. Vitamin D-refractory rickets and cystine storage disease in a child. (*A*) Evidence of rickets is manifested by thick epiphyseal cartilage as seen in only the distal end of the femur. The patient had severe renal glomerular damage in addition to the renal tubular defect, producing increased density of the bones and subperiosteal resorption. This is not well reproduced but in *B*, the tufts of the distal phalanges of the second, fourth, and fifth fingers show evidence of subperiosteal resorption. The medial aspect of the proximal end of the tibia also showed subperiosteal resorption.

ter of the bones in these patients was usually wider than normal. The cortex of the shafts in some regions was thick and had lost the homogeneous density normally seen in the compact bone. It was thin in the metaphyseal regions where thick trabeculae abounded (Fig. 7).

The calvarium was thick, and frequently, hyperostosis adjacent to the inner table was present.

We assume that the increased density is the result of over-abundance of bone matrix which is incompletely calcified. The total amount of calcium salts, however, exceeds that in normal bones which, although having less matrix, are well calcified. It has been demonstrated that in osteomalacia bone continues to be deposited but the trabeculae with uncalcified osteoid borders are resistant to resorption and thus tend to become thick.[23]

The simple tubular insufficiencies are frequently mild, not becoming clinically perceptible until late in childhood or until adult life.[10] The mineral depletion may be sufficient to stunt growth and produce bending deformities, but the over-abundance of bone matrix tends to compensate for structural inadequacies. Pseudofractures may occur in patients with increased or decreased bone density. Pseudofractures are not common in any type of rickets.

Accessory ossicles about joints, and exostotic growths and spurs at tendinous and ligamentous insertions are common in only this form of osteomalacia (Fig. 8, *A* and *B*; and 9).

These ossicles and exostotic growths were seen in 5 of 7 adults examined. The accessory ossicles were most frequent in the space adjacent to the radius, ulna, and carpal bones, but did occur in other joints, often associated with articular cartilage narrowing.[21] Similar ossicles about the wrists were found by Finby and Bearn[13] in 9 of 20 patients with hyperphosphaturia and osteomalacia associated with Wilson's disease. In our study, these lesions were not present in young children. In 1 child who was followed for several years, they developed after the ulnar and radial epiphyses had fused. Small spurs developed from the

radius which later became detached. Most likely, the spurs and exostotic growths are the result of stress to excessively active bone that has become weakened by depleted mineral stores.[5] Fairbank[12] observed that in resistant or continued rickets, exostotic growths of the medial surfaces of the upper tibia are more frequent than in ordinary rickets. Premature synostosis of the sagittal suture was present in 2 adults and 2 children. The synostosis was found on routine roentgen examinations of the skeleton and did not produce symptoms.

The more extensive renal tubular defects were seen only in children. Excess excretion of amino acids, protein, fixed base, and in 1 case, cystine storage (Debré-deToni-Fanconi syndrome) were associated with these defects. Dense bones, joint ossicles, or localized overgrowths were not present (Fig. 16, *A* and *B*).

III. Chronic Renal Glomerular Failure. The skeletal roentgenograms of children only were evaluated. Their lesions consisted of two separate entities, rickets and osteitis fibrosa. Except for 2 patients, all with unfused epiphyses had rickets and all but 2 of the 11 had hyperparathyroid bone disease (osteitis fibrosa). One patient had pseudofractures. In these respects they were similar to some patients with vitamin D deficiency, or malabsorption rickets, or osteomalacia. The 11 patients with renal failure had a greater propensity toward hyperparathyroid bone lesions than those with other forms of rickets or osteomalacia. When present, the lesions tended to be more advanced. Also, the bones varied in density, particularly in the metaphyses of the long bones, being sclerotic in some areas and demineralized in other adjacent areas. This is the only form of osteomalacia or rickets in which this irregular mineralization is seen. The sclerotic lesions were not symmetric. The vertebral bodies in some cases showed similar changes with the upper and lower portions presenting horizontal bands of increased density and the central part being relatively radiolucent. Occasionally, rickets alone was an early finding in renal glomerular failure. As the kidney disease progressed, roentgen signs of rickets subsided while signs of secondary hyperparathyroidism occurred or progressed. On autopsy studies of patients with renal disease, Gilmour[16] found that in the 3 in whom parathyroid hypertrophy was greatest, the rickets in both bone and cartilage had healed; however, the deformities of endochondral ossification which had formed while the rickets was active had remained. In 2 other patients with less hypertrophy, the rickets had remained active in the bone but healed in the cartilage. The microscopic sections of the bones of all 5 patients showed evidence of osteitis fibrosa. Gilmour observed that as the glands enlarged, rickets which had been active in both bone and cartilage became healed first in cartilage and finally in bone.

Metastatic calcification of the soft tissues, excluding the kidneys, was present in only 3 cases. It involved vessels and other soft tissues, particularly about joints in all 3, and the anterior ends of the ribs in 1. Calcification of the soft tissues is always associated with marked hypertrophy of the parathyroids and with osteitis fibrosa. Gilmour stated that the available evidence suggests that when renal osteodystrophy is associated with metastatic calcification, the rickets is healed. This was not completely true in 1 of our patients who had extensive soft tissue calcification and slight residual signs of rickets.[18]

In 1 patient, calcification of the vessels decreased as did the subperiosteal resorption of the phalanges when the patient was placed on a low phosphate diet. Subsequently, the vascular calcifications decreased further, but subperiosteal resorption returned. Growth and maturation were retarded in most patients, with growth being the more severely affected. In 1 patient with normal maturation, the tubular bones of the hands were extremely short.

The etiology of the increased density of the skeleton in osteomalacia has provided considerable discussion in the literature. The density that occurs in simple vitamin

D-resistant rickets or vitamin D-resistant osteomalacia is readily explained on the basis of excess bone which more than compensates for the incomplete calcification. In these cases, the density of the skeletal bone is relatively uniform.

In chronic renal glomerular failure, two mechanisms may be responsible. (1) In some patients, mainly children, spongy bone shows patchy areas of increased density. These lesions are not symmetric and are associated with subperiosteal resorption of the phalanges or other evidence of hyperparathyroidism. At necropsy or biopsy, hyperplastic parathyroid glands are present. Roentgenologically, the lesions are similar to those in primary hyperparathyroidism that have healed after surgical removal of a parathyroid adenoma, or in rare cases that present sclerotic lesions.[2,3,11] The effect of parathyroid hormone upon bone is primarily due to replacement of bone by osteoclasts, vascular fibrous tissue, and osteoid with varying calcification. Usually, destruction predominates so that on roentgenograms the effects of destruction are visible. If the adenoma is removed, the destructive lesions heal with an over-production of bone, presenting dense areas roentgenographically. Rats treated with parathyroid hormone also initially show radiolucent lesions, primarily in the metaphyses, but with continued treatment these lesions become more dense than normal bone. The lesions in hyperparathyroidism are usually not uniformly distributed, which is true also of the patchy lesions in some cases of chronic glomerular failure. It would thus seem likely that the irregular sclerotic lesions of chronic renal disease represent secondary hyperparathyroidism in which the healing phase predominates. The bones in these cases do show evidence of subperiosteal resorption, indicating that destruction still predominates in some cortical areas. Similar sclerotic lesions are seen in various blood dyscrasias associated with myelofibrosis, although the typical dense horizontal bands at the upper and lower margins of the vertebral bodies are absent.

(2) Other patients with glomerular failure, usually adults, show a uniform sclerosis of the skeleton.[7] The roentgenograms of these patients do not demonstrate evidence of hyperparathyroid bone disease and at necropsy the parathyroids are not enlarged. No transverse bands of the vertebral bodies are seen. The sclerosis in these patients appears to be the result of healed osteomalacia. It resembles in some respects the dense bones seen in osteomalacia due to renal tubular insufficiency. It is also possible that myelofibrosis with osseous metaplasia may be a factor. In some instances, as is frequently true in myelofibrosis, the increase in density is limited to the spine and pelvis. Myelofibrosis may result from the inhalation or ingestion of toxic chemicals (benzine, etc.), or be associated with leukemia or anemia. In chronic glomerular failure, the retained by-products of metabolism may be considered toxic to the tissues, including the bone marrow, and all of these patients have anemia of varying severity.

Other unexplained features of chronic glomerular failure previously described, but not reviewed in this study, are overgrowth of the mandible and maxilla,[14] and elongation of the extremities in relation to the trunk.[7]

The metabolic error in vitamin D-resistant rickets (renal tubular insufficiency) or in osteomalacia has been investigated extensively. Albright and Reifenstein[4] expressed the belief that it was caused by faulty intestinal absorption of calcium and that the phosphaturia was the result of secondary hyperparathyroidism. Dent[9] was of the opinion that a primary loss of phosphorus in the urine was due to decreased renal tubular resorption. Dent and Harris,[10] however, have recently agreed that some defect of intestinal absorption must also be present in addition to the tubular defect. Jackson and co-workers,[17] in reviewing both theories, stated that Albright and Reifenstein's was the more plausible, although more than one variety of resistant

osteomalacia may exist. Parathyroid hyperplasia, found occasionally on exploration, is difficult to reconcile with the roentgen findings in the patients of this study and in others who were seen but not included. In our experience, roentgenograms showed no signs of osteitis fibrosa in patients with vitamin D-resistant osteomalacia or rickets whose renal glomerular function was normal. In vitamin D deficiency with chronic renal failure, however, osteitis fibrosa was frequent.

SUMMARY

Osteomalacia is a disorder of bone tissue in which calcium salts have failed to deposit promptly in the formed bone matrix. In growing children, additionally impaired calcification of maturing cartilage at the zone of provisional calcification causes retarded endochondral ossification, known as rickets.

Osteomalacia or rickets presents 3 distinct roentgen patterns.

1. *Malabsorption and Dietary Deficiencies.* This group usually shows the classic decreased density of the skeleton involving both the peripheral and axial portions as well as the cortex and spongiosa. About half of the patients in this group have pseudofractures. In 5 of our 17 adults, roentgenographic signs of hyperparathyroidism were present, due to secondary hyperfunction of the parathyroid glands.

2. *Renal Tubular Insufficiency (Vitamin D-Resistant Osteomalacia or Rickets).* The simple, mild type with the resorption defect limited to phosphate and sometimes to glucose is the most common. Density of the bones in more than half of the adults and children was increased rather than decreased. The bones in these cases of increased density were usually wider and the diaphyseal cortex of the bones thicker than normal. Accessory ossicles about the joints and exostotic growths and spurs were common in only this form of osteomalacia but were not seen in children. In patients with more extensive renal tubular defects associated with excess excretion of amino acids, protein and fixed base, no dense bones, joint ossicles, or localized outgrowths were present.

3. *Chronic Renal Glomerular Failure.* Only the children in this group were evaluated. Their lesions consisted of two separate entities—rickets and osteitis fibrosa. Except for 2 patients, all with unfused epiphyses had rickets and all but 2 of the 11 patients studied had hyperparathyroid bone disease (osteitis fibrosa). In patients with renal failure, the propensity toward hyperparathyroid bone lesions was greater than in those with other forms of rickets or osteomalacia. The bones frequently varied in density, particularly in the metaphysis of long bones. This irregular mineralization is seen only in this form of osteomalacia or rickets. The vertebral bodies in some patients showed similar changes with the upper and lower portions presenting horizontal bands of increased density. Occasionally, rickets was the only early finding. As the kidney disease progressed, roentgen signs of rickets subsided while signs of secondary hyperparathyroidism appeared or progressed.

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OSTEOGENESIS IMPERFECTA IN THE ADULT*

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OSTEOGENESIS imperfecta is a hereditary congenital abnormality characterized in its classic expression by fragile bones, blue sclerae and otosclerosis. These manifestations may appear singly, in combination or may be undetectable by clinical examination. A considerable number of those affected die in infancy or childhood but many live to an advanced age. It is the purpose of this report to call attention to the adult with osteogenesis imperfecta.

At the present time there is no histologic or chemical procedure[1,9] by which a diagnosis of osteogenesis imperfecta can be established. Microscopic sections of these bones show osteoporosis[1,5,6] and the urine contains decreased quantities of hydroxy-L-proline[13] but these are nonspecific abnormalities occurring in other diseases. Osteogenesis imperfecta may be diagnosed by its classic pattern of hereditary transmission[10] of one or more elements of the syndrome or by the occurrence of multiple fractures without adequate trauma dating back to infancy.[3,4] These fractures may occur in bones which appear normal roentgenographically or are more often attenuated and undermineralized. No other disease shows this tendency to fracture.

In the following 11 cases of adult osteogenesis imperfecta, the diagnosis was established as follows. In 10 cases there was hereditary transmission of one or more elements of the three cardinal traits of the syndrome. In 1 case without known familial history there were multiple fractures without adequate trauma. This last case probably represents a mutation or a phenocopy and the distinction may never be made.

REPORT OF CASES

CASE I. M.C. is a 60 year old self-employed male with body habitus of average proportions.

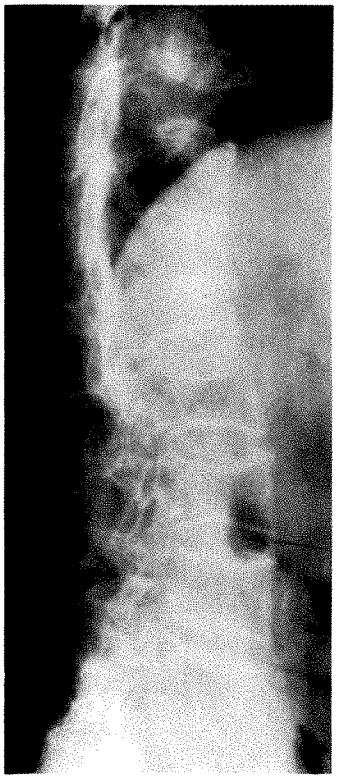


FIG. 1. Case 1. Severely hypomineralized dorsolumbar spine with biconcave vertebral bodies.

He has blue sclerae, impaired hearing and has had approximately 100 fractures. There is minimal limitation of physical activity. Extensive metabolic studies added no significant information to the diagnosis of osteogenesis imperfecta.

Permission for examination was limited to the vertebrae, chest and pelvis. There is severe hypomineralization resulting in poor roentgenographic contrast. Minimal deformity is present at the sites of well healed fractures and there is protrusio acetabuli. The vertebral bodies are biconcave (Fig. 1) and there is severe scoliosis.

CASE II. J.C. is a 27 year old school teacher, the son of M.C. He has blue sclerae and there have been at least 30 fractures, all healing without deformity and all occurring following minor

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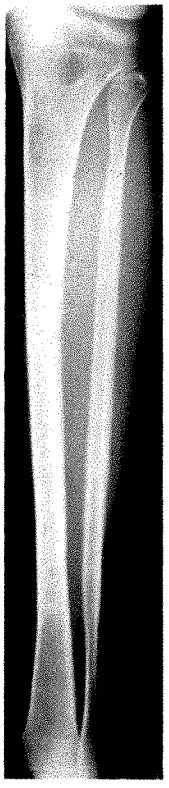


Fig. 2. Case II. Long straight slender tibia and fibula with numerous growth lines. Poor trabeculation and slight hypomineralization are demonstrated.

trauma. He is tall and of average physique. The bones are somewhat slender (Fig. 2) and many show well healed fractures with slight deformity. There is poor mineralization of the spine with minimal biconcavity of the vertebral bodies. The lateral roentgenogram of the skull shows platybasia and huge frontal, sphenoid and mastoid sinuses (Fig. 3). Dentinogenesis imperfecta[7] is severe.

Case III. E.O., the propositus in this family grouping, is a 53 year old housewife with blue sclerae and a history of multiple fractures. Her father lived to an advanced age with brittle bones. She required hospitalization because of severe pain in the right foot. Roentgenograms of the foot demonstrated a pattern compatible with atrophy of disuse but no fractures were seen. There is severe generalized hypomineralization of bone with biconcave vertebral bodies but the long bones show normal dimensions. A lateral projection of the skull shows platybasia.

Case IV. J.P. is the 48 year old unmarried self-employed brother of E.O. He is of average build, wears a hearing aid and has had at least 20 fractures. The thoracic vertebral bodies show compression fractures and are undermineralized (Fig. 4) while the lumbar bodies have a biconcave shape. The sphenoid, frontal and mastoid sinuses are unusually large and there is platybasia. An unsuspected fresh fracture of the olecranon process[10] (Fig. 5) was discovered during the bone survey. The olecranon process was removed and on microscopic examination showed only osteoporosis. All the long bones have normal dimensions with hypomineralization and a decrease in the prominence of the trabecular pattern.

Case V. M.K. is the 36 year old married daughter of E.O. She has blue sclerae but states she has had no fractures. A skeletal survey demonstrated only a slight scoliosis and all bones appeared normal in dimensions, configuration and mineralization. Her only child has blue sclerae and has had two fractures.

Case VI. M.D. is a 27 year old married female of average size. She has blue sclerae, a hearing deficit and has had approximately 6 fractures. There is an antecedent history of fragile bones and her older brother has blue sclerae but she does not know whether he has had fractures. A 10 year old son with blue sclerae has had two fractures but these may have occurred following adequate trauma. Her skeletal survey shows slender normally proportioned bones with aver-

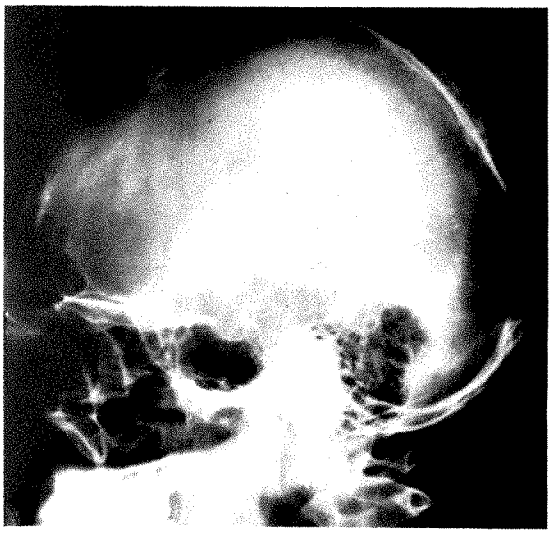


Fig. 3. Case II. Excessively large frontal, sphenoid and mastoid sinuses.

age mineral content. There is a slight dorsal scoliosis with a minimal compression fracture of the sixth dorsal vertebra.

Case vii. A.G. is a 33 year old married female of average size who has had about 35 fractures. There is an antecedent history of fragile bones and her daughter also has osteogenesis imperfecta. The long bones are of average dimensions and mineral content but there is slight lateral bowing of the femurs and medial bowing of the tibias and fibulas. The skull shows platybasia and large frontal sphenoid and mastoid sinuses. A severe rotatory scoliosis (Fig. 6) prevents adequate study of the individual vertebral bodies.

Case viii. F.B. is a 33 year old married male, taller than average and with a heavy build. There is no known parental history of fractures but a daughter has osteogenesis imperfecta. He has had about 25 fractures, blue sclerae, and has impaired hearing. Roentgenographic studies of the spine and skull only were permitted.

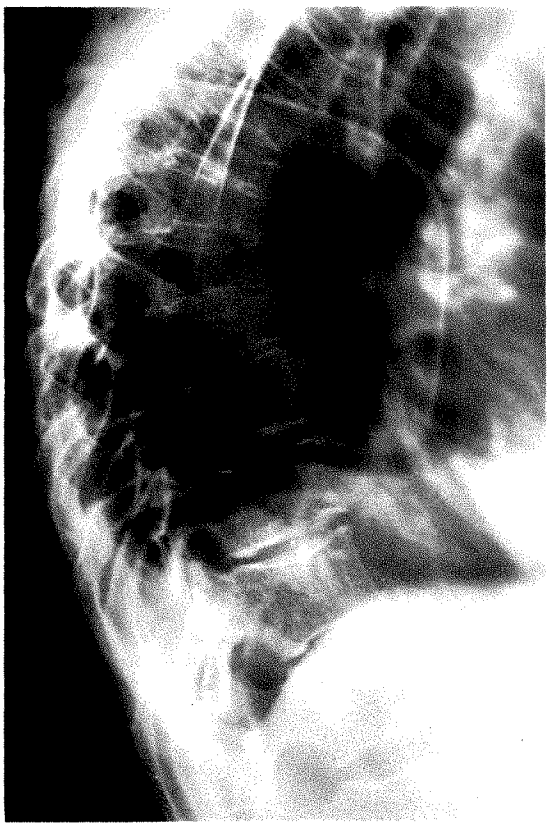


Fig. 4. Case iv. Kyphotic hypomineralized dorsal spine with multiple compression fractures.



Fig. 5. Case iv. Recent fracture of the olecranon process, probably an avulsion fracture. Note the normal osseous structure.

There are no fractures demonstrable and the vertebrae appear normal. The skull shows large frontal, sphenoid and mastoid sinuses and platybasia (Fig. 7).

Case ix. W.S. is a 20 year old unmarried male of average height and husky build who has had more than 20 fractures without adequate trauma. There is no known familial history of fragile bones. A roentgenographic survey shows localized scoliosis of the upper dorsal spine, the lumbar vertebrae show a slight biconcavity and the roentgenograms convey a distinct impression of osseous hypomineralization. Both femurs (Fig. 8) show moderately severe lateral bowing but the remainder of the long bones appear to have normal contours, dimensions and mineralization. A lateral roentgenogram of the skull shows large sphenoid, mastoid and frontal sinuses. There is a recent central acetabular fracture with comminution of fragments.

Comment. This patient has no familial history of the fragile bone syndrome and he has no descendents. In the absence of a genetic background, this case probably represents a mutation. Idiopathic osteoporosis[6,8] is an unlikely consideration in this patient since fractures occurred early in childhood, while fractures in idopathic osteoporosis occur after the age of 20 years and are usually confined to the spine.

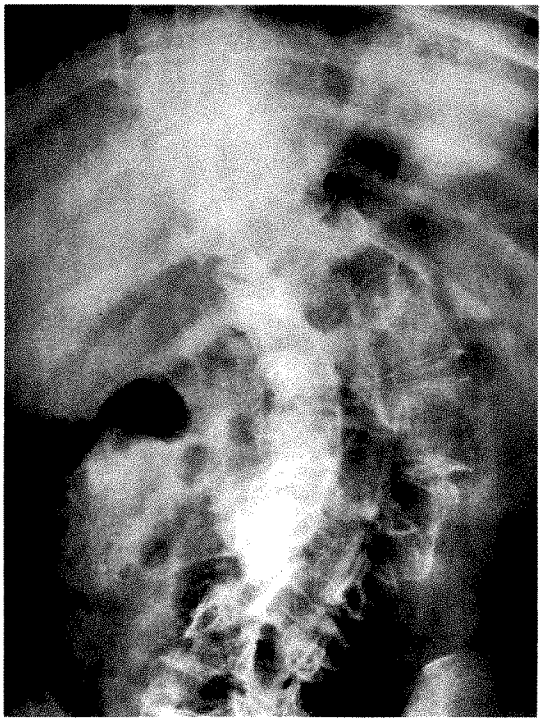


Fig. 6. Case VII. Severe rotatory scoliosis of lumbar spine. The bodies are severely demineralized.

Case X. L.J. is an adult female with a familial history of osteogenesis imperfecta but other details of the history are not obtainable. Roentgenograms of an extensive skeletal survey show severe bilateral protrusio acetabuli with compression fractures of the third and fifth lumbar vertebrae. The right humerus shows a fracture deformity, there are well healed fractures of the left wrist and there is an ununited fracture of the left carpal navicular. Segmental fractures with nonunion and overriding of fragments are present in the right femur and there is severe bowing and nonunion of a transverse fracture of the left femur (Fig. 9).

In those areas where severe fracture deformity has not interfered with function, the bones have normal dimension and mineral content.

Case XI. F.M. is an adult male with a familial history of osteogenesis imperfecta but further information is unavailable. There are multiple compression fractures in a severely kyphoscoliotic spine and the pelvis has a triradiate configuration. The ribs, a fibula and a radius are severely attenuated and there are fracture deformities of the femur. The skull is normal in shape. The architectural pattern and the dimensions of the less severely affected bones are normal.

ROENTGEN FEATURES

Osteogenesis imperfecta in the adult has no pathognomonic roentgen findings but there are highly suggestive features. Multiple fractures of the long bones in osseous tissues which are roentgenographically normal except for minimal to moderate degrees of undermineralization is presumptive evidence for the diagnosis of osteogenesis imperfecta. This diagnosis is virtually a certainty when fractures occur in the absence of significant trauma. Biconcave vertebral bodies as well as compression fractures are also a frequent finding and severe scoliosis may be seen with or without intrinsic vertebral abnormality. All fractures heal with the same degree of rapidity as in normal bones and without an increased incidence of complications. There was no example of hyperplastic callus formation seen in adults.

Platybasia is known to be a frequent occurrence in osteogenesis imperfecta and was found in 4 cases. A feature which is at least as frequent but which has never been reported is the presence of unusually large paranasal and mastoid sinuses. In 5 of these

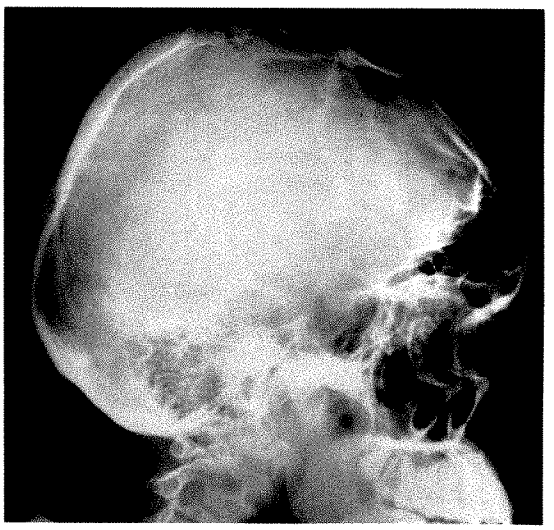


Fig. 7. Case VIII. Unusually large frontal, sphenoid and mastoid sinuses. There is platybasia.

11 cases the sinuses were as large as those expected to be found in acromegaly.

All patients were more than 155 cm. tall except Cases x and xi, where the height was unknown. In these last 2 cases the bones were attenuated only where fractures and deformity were severe; where there was little or no deformity the bones had normal diameters. In the first 9 cases all bones were within the normal range of size and the cortex appeared to be of normal thickness. There was, however, a distinct impression conveyed of hypomineralization and a decrease in the trabecular pattern of most bones.

DISCUSSION

In virtually every textbook other than in those genetically oriented, osteogenesis imperfecta is treated as a group of closely related diseases. Within the past few years, geneticists have demonstrated that these syndromes with multiple clinical and eponymic designations are only varied manifestations of a single disease.[1,10] They have shown that osteogenesis imperfecta is a non-sex linked autosomal dominant hereditary disease[13] with high penetrance and wide expressivity. The only deviation from this hereditary pattern is the rare occurrence of *de novo* mutations. As a rule these cases are severe and the patients die in the neonatal period.[2] Those who survive may establish a new lineage also with a great variability of expression.



Fig. 8. Case ix. Lateral bowing of both femurs. The osseous structures appear normal.

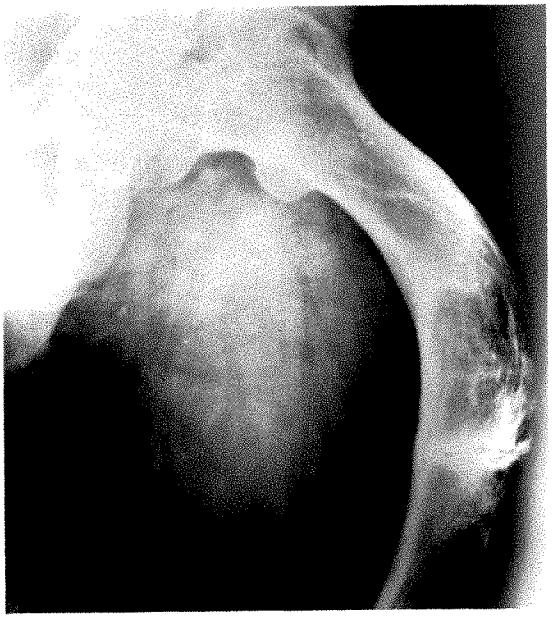


Fig. 9. Case x. Severe lateral bowing of femur with nonunion of a transverse fracture.

Extensive analysis of the karyotype[12] in osteogenesis imperfecta has not shown abnormality. It is believed that the complete galaxy of expression of this disease is the result of a defect in a single gene controlling the transformation of immature collagen or reticulin to mature connective tissue.[5,10,13] It has not been demonstrated whether this is a qualitative or quantitative defect.[13] The defective collagen supplies "inadequate" bone matrix[6] resulting in fragile bone; ligaments and tendons are structurally weak resulting in sprains;[15] the scleral tissues are thin and translucent allowing the blue coloration of the choroid to show; and scoliosis may develop either as a result of laxity of spinal ligaments or of vertebral fractures. It is also of interest that the only known chemical aberration is the decreased urinary excretion of hydroxy-L-proline and

all other urinary amino acids are unaffected. Hydroxy-L-proline[18] is present in large amounts in the collagen protein molecule and in experimental scurvy with osteoporosis there is also a decrease in the urinary excretion of this amino acid.

The variations in penetrance and expressivity are controlled by modifying genes[11,18] but the nature and number of these genes are unknown. Environmental factors[11] will also contribute to the severity of presentation of the disease. The similarity in the genetic constitution or genotype of sibs results in less intrafamilial than extrafamilial variability of expression and in sibships there is a distinct tendency for similar degrees of severity of manifestation.

An interesting clinical facet of this problem is the relative insensitivity with which these patients react to their fractures.[10] At times, minor fractures may pass unnoticed as was the avulsion fracture of the olecranon process in Case IV. There is, however, no abnormality of the nervous system and the perception of all types of sensation including pain is unimpaired. Because of their comprehension and familiarity with fractures and the lack of the potentiating influences of fear and anxiety on pain, they cooperate remarkably well during diagnostic and therapeutic procedures.

CONCLUSION

The gloomy prognosis extended in cases of osteogenesis imperfecta is not an accurate portrayal of the natural evolution of the disease. A considerable but unknown proportion of those affected will survive to an advanced age, living a useful and productive life. Many will have no obvious stigmata of the disease and some may even escape detection. The number of fractures sustained may be as many as 100 as in Case 1 or there may never be a fracture. The possibility of osteogenesis imperfecta should be considered in any case where multiple fractures occur following minor trauma regardless of the roentgenographic appearance of osseous solidity. In these cases an inquiry into the genetic background is indicated.

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SKELETAL MATURATION RATES IN MONGOLISM*

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RELATIVELY precise methods of assessing skeletal maturity have been described by Flory[11] and Todd.[36] Standards of maturity for use in making assessments by these methods have been published by several workers of which the most widely used are those of Greulich and Pyle.[14] Despite the increasing and widespread use of these standards in the study of normal development, few investigations of skeletal maturation rates in disease have been reported. An analysis of skeletal maturation rates in mongolism has been made because it could provide valuable information relative to the nature, diagnosis and prognosis of this condition.

REVIEW OF LITERATURE

Carpal Ossification. Some authors report that carpal ossification is often advanced at birth[2,22] despite the short gestation period associated with this disease.[32] Some state that carpal ossification begins at a normal age in a majority of mongoloid children,[10,17,18,23] but others state that its commencement is delayed in a majority.[5,12,28,29,34,37] All of these statements are based upon the examination of small groups of mongoloids. A wide variation between individual mongoloids in age at onset of ossification has been reported[4,13,22,35] and, consequently, a large body of data must be examined before reliable conclusions can be drawn.

Epiphyseal Ossification. Holt[16] reported that epiphyseal ossification began at a normal or an early age in almost all mongoloids. Siegert,[30] however, reported that the commencement of epiphyseal ossification was delayed in most mongoloids.

Skeletal Maturation. There is evidence that skeletal age is similar to the chronological age in almost all mongoloids.[15,33] In the group of 16 mongoloids examined by Menghi,[20] retardation was more marked among the males than the females. It has been reported that, although a majority had a skeletal age within 2 s.d. of the mean for their chronological age, retardation beyond 2 s.d. was present in 44 per cent[20] and 21 per cent[9] of two groups of mongoloids. Studies of skeletal maturation rates in mongolism have not been reported previously.

MATERIAL AND METHODS

Standardized roentgenograms of the left hand-wrist areas of 149 mongoloid children (63 males, 86 females) of white Australian parentage were studied. All the mongoloid children included in this study lived in institutions except 7 males and 7 females, aged more than 6 years, who lived with their parents. Assessments were made of 353 roentgenograms of the males and 444 of the females. Each roentgenogram had been taken within one week of the chronological age to which it was assigned, *e.g.* all roentgenograms considered in the 6 year group were taken between the chronological ages of 5 years 11 months 3 weeks and 6 years 1 week. All the roentgenograms were made according to the instructions of Greulich and Pyle[14] and each was assessed using the Greulich-Pyle hand-wrist standards of skeletal maturation.[14] At least two serial roentgenograms of each child were assessed and the findings were analysed statistically. Some aspects of the recorded data were compared with findings concerning normal white Australian children obtained during a multi-discipline longitudinal study.[26,27]

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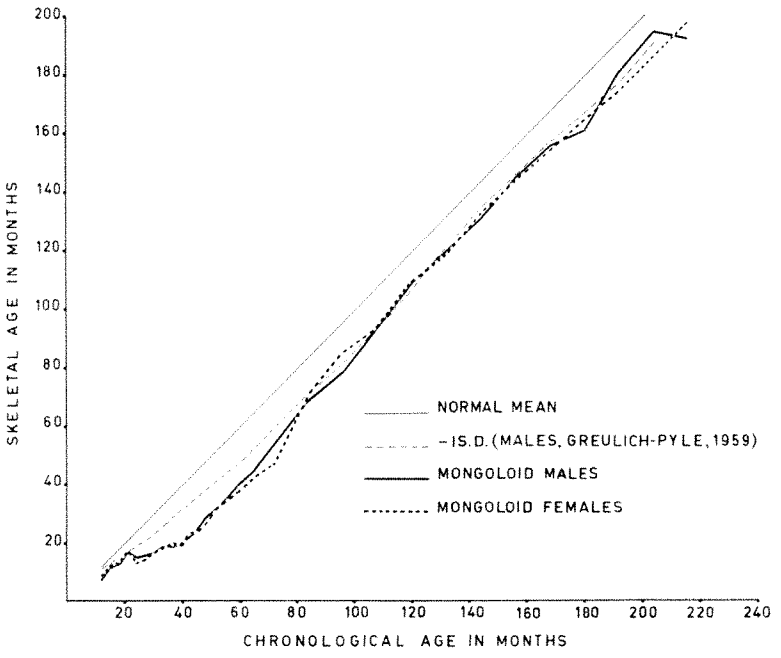


FIG. 1. A graph of skeletal age against chronological age for mongoloid males and females using all the recorded data in a cross-sectional manner. The theoretical mean and one standard deviation below the mean (males) have been included.

FINDINGS

CROSS-SECTIONAL ANALYSIS

Mean Skeletal Ages. The mean skeletal ages, at each chronological age considered, have been recorded in Table 1 and have been shown graphically in Figure 1. Reliable conclusions cannot be drawn from the data recorded at chronological ages less than 24 months because the sample sizes were small at those ages. At all ages studied, the sex differences in mean skeletal ages were small in the mongoloids and the mean skeletal ages were less than the corresponding chronological ages. The trend of the points plotted on this graph (Fig. 1), relating to mongoloids of each sex, can be represented reasonably by two straight lines that would meet at about the chronological age of 6 years. The lines representing the trend of the points for children aged more than 6 years are steeper than the corresponding lines for younger mongoloids.

In Figure 1, the level of 1 s.d. below the mean for normal males[14] has been represented. For clarity, the corresponding and closely similar level for normal females has been omitted. It can be seen that the mean skeletal ages of the mongoloids studied were close to 1 s.d. below the normal mean, except between the chronological ages of 2 and 7 years when the differences were greater. Despite these differences between the normal mean skeletal ages and the mean skeletal ages of the mongoloid groups, there were 7 male and 15 female mongoloids in whom, at one or more assessments, the skeletal age exceeded the chronological age. At all chronological ages, the standard deviations for mongoloid children were larger than those for normal children.

Skeletal Ages of Individual Bones. These were analysed statistically using one record of each mongoloid child who had been assessed between the chronological ages of 3 years 5 months and 4 years 7 months. For each of 18 male and 17 female mongoloids, the record was selected which had been made closest to the chronological age of 4 years. From these records, the mean differences between the skeletal ages of indi-

vidual bones and the mean skeletal ages on the same sides of the same children were calculated. Graphs based on these calculations are presented in Figures 2 and 3 for males and females, respectively. In these graphs, the mean differences between the skeletal ages of individual bones and the mean skeletal age on the same side of the same individuals have been recorded, together with the range of these differences (+1.5 s.e. to −1.5 s.e.). Data relating to the ulna have been omitted from these graphs because it is difficult to assign an accurate skeletal age to this bone, during the chronological age range being considered. Data relating to the capitate and hamate were included but assessments of other carpal bones were omitted because they could be made in only a few children and these particular children were accelerated in maturation compared with the remainder of the group. By inspection of these graphs, one can visualize the extent of the tendency for individual bones to vary from the mean skeletal age. It can be seen that, in the group of boys studied (Fig. 2), there was a tendency for the separate metacarpals and proximal phalanges to be less mature as bones progressively closer to the ulnar side of the hand were considered. Among the girls (Fig. 3), a similar tendency was noted in the metacarpals and in the proximal, middle and distal phalanges. In these graphs, if the intervals from +1.5 s.e. to −1.5 s.e. overlap for two different bones, the difference between the variations of the

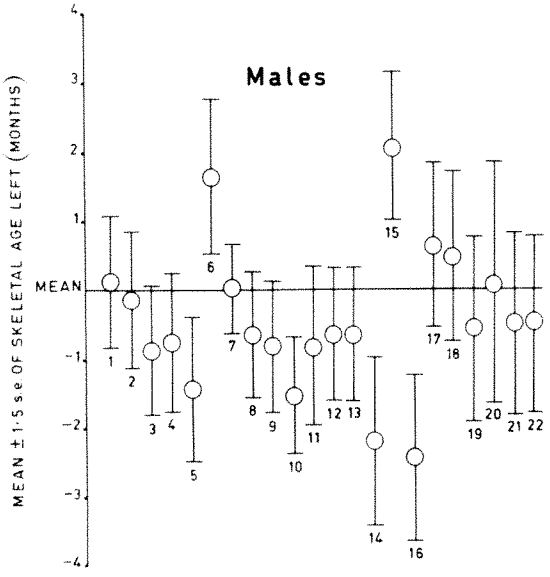


Fig. 2. The variation of the skeletal ages of individual bones from the mean skeletal ages on the same sides of the same male mongoloids. Each vertical line represents the mean (o) ± 1.5 s.e. The numbers indicate bones as follows:

1–5	Metacarpals	I–V
6–10	Proximal Phalanges	I–V
11–14	Middle Phalanges	II–V
15–19	Distal Phalanges	I–V
20	Radius	
21	Capitate	
22	Hamate	

Table I

MEAN SKELETAL AGES

Chronological Age Months	Males		Females	
	n	Mean Skeletal Age Months	n	Mean Skeletal Age Months
12	1	7.35	3	8.22
15	1	11.85	1	12.15
18	3	13.25	3	13.64
21	1	16.81	3	17.37
24	17	14.66	25	13.45
27	21	15.56	23	14.06
30	18	16.80	20	16.48
33	21	18.33	19	18.08
36	19	18.57	14	19.75
39	14	20.86	16	19.36
42	20	22.54	13	23.41
45	17	24.56	15	24.12
48	14	28.52	17	26.32
54	17	33.42	19	33.55
60	19	40.00	16	38.18
66	19	45.66	16	43.40
72	14	53.20	15	46.67
84	13	68.59	12	71.43
96	15	78.58	12	85.49
108	12	92.96	13	93.62
120	8	108.51	13	108.81
132	7	119.31	17	118.93
144	8	130.98	17	132.98
156	9	144.80	28	144.06
168	12	155.71	29	154.36
180	10	161.14	26	165.16
192	12	180.68	21	175.00
204	8	195.32	13	187.01
216	3	192.20	5	198.88

n = number.

TABLE II

THE MEAN SLOPES OF SKELETAL AGE AGAINST CHRONOLOGICAL AGE IN GRAPHS FOR INDIVIDUAL MONGOLOID AND "NORMAL" CHILDREN

	Males					
	C.A. 0–6 Years			C.A. 6+ Years		
	n	$\bar{b}$	s.d.	n	$\bar{b}$	s.d.
Mongoloid Children						
2–4 Assessments	13	0.499	0.310	14	0.963	0.376
5 or more Assessments	14	0.597	0.298	2	1.376	0.043
Living at Home	—	—	—	7	1.088	0.295
"Normal" Children	28	0.956	0.080	—	—	—

	Females					
	C.A. 0–6 Years			C.A. 6+ Years		
	n	$\bar{b}$	s.d.	n	$\bar{b}$	s.d.
Mongoloid Children						
2–4 Assessments	15	0.526	0.275	27	1.088	0.350
5 or more Assessments	15	0.637	0.261	12	0.989	0.293
Living at Home	—	—	—	7	1.019	0.192
"Normal" Children	27	0.973	0.066	—	—	—

C.A. = chronological age; $\bar{b}$ = mean slope; n = number; s.d. = standard deviation.

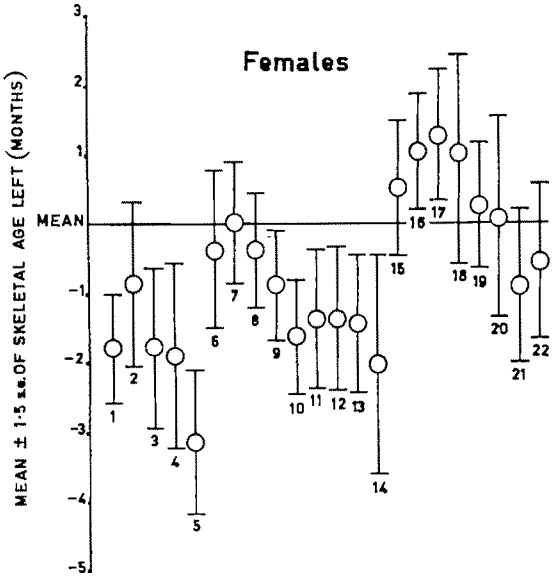


FIG. 3. The variation of the skeletal ages of individual bones from the mean skeletal ages on the same sides of the same female mongoloids. Each vertical line represents the mean (o) ± 1.5 s.e. The numbers correspond to bones in the same way as in Figure 2.

skeletal ages of these bones from the mean skeletal age on the same side of the same individuals is not statistically significant at the 5 per cent level. For example, in the group of mongoloid boys, the variation of the skeletal age of the first metacarpal from the mean skeletal age on the same side of the same individuals is significantly different statistically from the corresponding variations of the fifth middle phalanx and the first and second distal phalanges.

LONGITUDINAL ANALYSIS

A graph was drawn to an arithmetic scale, of skeletal age against chronological age for each mongoloid child. In all the graphs, the trend of the plotted points could be represented reasonably by a straight line. The slight deviations from linearity that were present did not occur systematically in a particular direction. The slopes of the lines used to represent the points on these graphs were calculated.

These slopes indicated the amount of change in skeletal age per unit of chronological age (skeletal maturation rate). The means and standard deviations of these slopes have been recorded in Table II and the results of significance tests relative to these data have been recorded in Table III. Many of the findings that follow are based upon an analysis of these slopes that correspond biologically to skeletal maturation rate.

Data derived from the mean slopes of mongoloid children have been separated depending on whether they were derived from 2–4 assessments or 5 or more assessments (Table II). In both the age groups considered and in both males and females, the mean differences associated with differences in the number of assessments were small and they were not statistically significant (Table III). The relative sizes of the means and standard deviations indicated that the variances of the slopes of the mongoloids assessed 2–4 times were similar to those of the mongoloids assessed 5 or more times.

For both male and female mongoloids, the mean slopes were steeper in the group with chronological ages exceeding 6 years than they were in the younger group (Table II). These differences were not due to selection by death. The results of the "t" tests (Table III) showed that these differences were statistically significant at the 1 per cent level for both sexes. In calculating these "t" tests, the mongoloid data were separated according to sex but those derived from 2–4 assessments were pooled with those derived from 5 or more assessments because there were no real differences between the mean slopes derived from them. The mean slopes and their standard deviations, for the mongoloid children (13 males, 10 females) whose serial records spanned the chronological age of 6 years, have been omitted from Table II. The values of the means were intermediate between those of the younger and older mongoloid groups. The mean slopes of the mongoloid children living with their parents were similar to the means for mongoloid children living in institutions (Table II) and the differences between them were not statistically significant (Table III).

The mean slopes of the mongoloid children who had been assessed 5 or more times were compared with those for "normal" Australian children using unpublished data[26] for the chronological age range 0–6 years. It was assumed that the linear nature of the individual graphs of skeletal age against chronological age for "normal" children up to the chronological age of 6 years would have continued at later ages and that the slopes would not have altered. The mean slopes of the mongoloid males

TABLE III

SUMMARY OF TESTS FOR SIGNIFICANCE OF DIFFERENCES BETWEEN MEAN SLOPES ON GRAPHS OF SKELETAL AGE AGAINST CHRONOLOGICAL AGE

Comparison	Males		Females	
	t	d.f.	t	d.f.
A	4.44**	14	4.91**	15+
B	0.84	25	1.13	28
C	1.51	14	0.85	37
D	4.44**	41	6.39**	67
E	0.45	21	0.19	44+
F	0.61	15	1.55	43

A = M. 5 or more assessments and C.A. 0–6 years versus
N. 5 or more assessments and C.A. 0–6 years.
B = M. 2–4 assessments and C.A. 0–6 years versus
M. 5 or more assessments and C.A. 0–6 years.
C = M. 2–4 assessments and C.A. 6+ years versus
M. 5 or more assessments and C.A. 6+ years.
D = M. with C.A. 0–6 years versus
M. with C.A. 6+ years.
E = M. living at home and C.A. 6+ years versus
M. that remain with C.A. 6+ years.
F = M. 6+ years versus
N. 6+ years.

$$t = \frac{\text{difference in sample means}}{\sqrt{S^2\left[\frac{1}{n_1} + \frac{1}{n_2}\right]}}$$ where S^2 is the pooled variance

from the two samples and n_1 and n_2 are the two sample sizes; d.f. = degrees of freedom; M. = mongoloids; N. = "normal" children; C.A. = chronological age; ** = significant at the 1% level; + = calculated according to the method for unequal variances (see BAILEY, N. T. J., Statistical Methods in Biology. John Wiley and Sons, New York, 1959).

and females were flatter than those of corresponding "normal" children (Table II). The differences between the mean slopes of the mongoloid and the "normal" children were statistically significant at the 1 per cent level, for both males and females in the younger age groups; the differences between the older age groups were not statistically significant (Table III).

For chronological ages up to 6 years, the variances of the slopes of the graphs for individual mongoloid children were greater than the corresponding variances for "normal" children. Six mongoloid children had steeper slopes, on their graphs of skeletal age against chronological age, than the slopes of any of the "normal" children.[26] An examination of the clinical, anthropometric and psychological records of these 6 children did not reveal any unusual features that would account for the steepness of their slopes.

CONCLUSION

The mean skeletal ages of the mongoloids who were studied during the present investigation were about 1 s.d. below the mean for "normal" children, except between the chronological ages of 2 years and 7 years when greater retardation was noted. The retarded skeletal maturity status of these children reflects their slow mean rates of skeletal maturation. These rates were slower than those of "normal" children to an extent that was statistically significant for the group aged 0–6 years but not for the older group. It is difficult to reconcile these findings with reports that, in most mongoloids, ossification of carpal bones commenced at a normal age,[10,17,18,23] that epiphyses began to ossify either at a normal or an early age[16] and that the skeletal age was similar to the chronological age.[15,33] The findings reported here do not confirm the sex difference in skeletal retardation noted by Menghi[20] in 16 assessments of skeletal age in mongoloids.

The cross-sectional and the longitudinal analyses provide evidence that skeletal maturation proceeds at a slower rate in mongoloids between the chronological ages of 2 years and 6 years than in older mongoloids. The differences between the rates of skeletal maturation of the two groups are highly significant statistically. There is suggestive evidence in the report of Benda[3] that corresponding changes might have been present in his material.

It has been shown that, in "normal" children, differences in levels of calcium intake and differences in illness experience were not related to the rate of skeletal maturation,[7] although "malnutrition" may be associated with skeletal retardation.[8] Assessments of the dietary intakes of many of the mongoloid children studied[6] indicated that their levels of intake of essential nutrients were similar to those of "normal" children. These observations, in addition to the fact that there were no real differences between the mean rates of skeletal maturation of mongoloids living at home and those of mongoloids living in institutions, make it unlikely that dietary factors were responsible for the skeletal retardation that was noted.

The differences between the mean rates of skeletal maturation of mongoloids who were assessed 2–4 times and the mean rates of skeletal maturation of those who were assessed 5 or more times were not statistically significant. Commonly, an individual child was assessed only a few times because the child was ill with minor infections when some assessments were scheduled. The findings reported by Cahn and Roche,[7] concerning the influence of illness on skeletal maturation rates in "normal" children, would lead one to expect the findings that were observed during the present study.

In "normal" children the rate of skeletal maturation is closely associated with the chronological age of attainment of adult stature.[1] The author has been unable to find reported data concerning the chronological age at which mongoloids cease to increase in stature, but Benda[3] has stated that, in mongoloids, the fusion of epiphyses to shafts occurs at a younger age than in nor-

mal children. This would not be expected from a consideration of the findings reported in the present paper.

A close relationship between the rate of maturation of the reproductive system and the rate of skeletal maturation is well established in "normal" children.[31] The findings reported in the present paper would lead one to expect that menarche would be delayed in mongoloid girls. Such a delay has been reported,[3,4,21] but Engler[10] has stated that menarche occurs at a normal age in mongoloid girls. It should be noted that an early onset of the menopause has been reported for mongoloids.[3,10]

Marked variations were noted within the mongoloid group studied. There were some mongoloids in whom the skeletal age was in excess of the chronological age despite the fact that the mean skeletal age for the group was markedly less than the corresponding chronological age. The skeletal maturation rates were more variable in the mongoloid children than in "normal" children and there were some mongoloid children with more rapid rates than any of the "normal" children. Some of this variability, within the group of mongoloids, might have been due to differences between individual children in the severity of the disease.

The differences between the skeletal ages of individual bones and the mean skeletal age, on the same side of the same mongoloids, can be compared with corresponding data for "normal" children of similar chronological age and the same racial origin.[25] The mean variations were smaller in the mongoloid males than in the "normal" males but no such differences were noted between the two groups of females. In both males and females, the s.e. of the differences were larger in the group of mongoloids than in the "normal" group for the majority of the bones studied. The tendency in the mongoloids for bones near the ulnar side of the hand to be less mature than others was present, but was less marked, in the "normal" children. A comparison of corresponding individual bones in the mongoloids and in the "normal" children showed wide differences between the two groups in the tendencies of particular bones to vary from the mean skeletal age on the same sides of the same individuals. The differences between mongoloid and "normal" children of the same sex in this regard are much greater than the sex differences present in the groups of mongoloid and "normal" children.

In mongoloid children of both sexes, the mean skeletal age of the fifth middle phalanx was less than the mean skeletal ages on the same sides of the same individuals. The fact that similar findings have been reported for "normal" children[25] makes it unlikely that the relative skeletal retardation of this bone is associated with the fact that it is commonly hypoplastic in mongoloids.[24] The report of Bullard[5] that the carpal and forearm bones were more retarded than the other bones of the hand-wrist area in mongoloids has not been confirmed by the present study.

The evidence relating to the hormonal changes associated with mongolism has been reviewed recently.[3,9] A consideration of the incomplete and sometimes conflicting nature of the information available makes it clear that the exact nature of these hormonal changes is still uncertain. The retardation of mean skeletal age and the slow mean rates of skeletal maturation reported in this paper should be considered in an analysis of the hormonal abnormalities associated with this condition. The finding that in some mongoloid children the skeletal age was advanced and that in some the rates of skeletal maturation were unusually rapid, makes it possible that the hormonal changes in mongolism might be as variable as the clinical signs.[19]

The diagnostic usefulness of the assessment of skeletal age in cases of possible mongolism must be judged on the basis of the cross-sectional findings. These findings revealed wide variations between individual mongoloids and such assessments are probably of no diagnostic value in relation to this disease. There was no indication, in the

recorded data; that the prognosis was worse in those mongoloids in whom the rate of skeletal maturation was very slow.

SUMMARY

1. Serial skeletal age assessments of 149 mongoloid children have been analysed.

2. The mean skeletal ages of the mongoloid children were about 1 s.d. below the mean for normal children at chronological ages greater than 7 years. Before the age of 7 years, the skeletal retardation was greater.

3. In the 0–6 years group, the mean rates of skeletal maturation were slower than in the "normal" children to a statistically significant extent. The differences between the mean rates of older mongoloids and "normal" children were not significant.

4. There were statistically significant differences between the mean skeletal maturation rates of mongoloids aged 0–6 years and those aged 6+ years.

5. The variances of skeletal maturation rates were greater in mongoloid children than in normal children.

6. The differences between the skeletal ages of individual bones and the mean skeletal age on the same sides of the same individuals have been compared with corresponding data for "normal" children.

7. The significance of these findings has been discussed.

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HYPEROSTOSIS OF THE VAULT OF THE SKULL IN ACROMEGALY*

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THE author's purpose is to present an analysis, from a morphologic point of view, of hyperostosis of the cranial vault in acromegaly with the object of: (a) describing the various characteristics concerning the structure of the skull, the site and the extent of the process; and (b) evaluating, if feasible, possible relationships with other changes found in the acromegalic skull.

Among the various changes in the skeleton that make up the typical picture of acromegaly, the modifications of the cranium have been the subject of numerous studies. In these descriptions, excepting the classic malformation of the sella turcica, which, moreover, is not always present,[4,5,12,13] attention is paid mostly to the facial bones, the mandible and the paranasal cavities. As regards the cranial vault, the description is usually limited to the possible presence of thickening, sometimes associated in advanced cases with generalized osteoporosis. Other authors[13] are of the opinion that the cranial vault is, for the most part, of normal thickness, and that skull abnormalities in acromegaly are, as far as can be ascertained roentgenographically, confined to its visceral portion.

More detailed descriptions of hyperostosis of the cranial vault in acromegaly are comparatively rare. Naito and Schüller[16] presented a case with hyperostosis of the entire calvaria, Cushing and Davidoff[4,5] described 1 case and Allison and co-workers[1] another. Henschen,[8,9] reporting on a short survey of the changes of the calvaria in acromegaly in 20 patients with a summary of 25 to 30 other cases—partly from personal observations and partly from the literature—found a change of the inner surface of the skull resembling hyperostosis frontalis interna in at least 14 cases, with a definite preponderance in females.

In 1938, Troell[18] reported 2 cases with the Morgagni syndrome complicated with acromegaly and thyrotoxicosis. Three other similar cases were reported in the same year by Junet.[11] From an anatomicopathologic standpoint, Junet concluded that in acromegaly associated with a goiter, the osteogenic and cartilaginous phenomena (with the exception of articular cartilage) are less pronounced than in acromegaly alone.[15] To the cases reported by these 2 authors, Moore,[14] in 1953, added 6 other personal cases which showed the coexistence of acromegaly, hyperostosis of the cranial vault, toxic goiter or simple thyrotoxicosis and diabetes mellitus (Table I). These combined findings, which he proposes calling the "Troell-Junet syndrome," are a fairly well-defined pathologic entity rather than a purely casual association.

Subsequently, in a recapitulatory monograph of his long study on hyperostosis cranii (1955), Moore[15] again analyzed the incidence of cranial hyperostosis in a fairly large group of acromegalic patients (Table II). He feels that a disorder of the pituitary gland may be the common source of acromegaly and idiopathic hyperostosis; that the two conditions may coexist, but that the pathogenesis of isolated idiopathic hyperostosis of the skull undoubtedly differs from that of the skeletal changes of acromegaly.

In this regard consideration should be given to: (a) the completely different clinical aspect of the two diseases; (b) the systemic changes of the skeleton as well as of the connective tissue in acromegaly, as compared to cranial hyperostosis, in which

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Table I

DESCRIPTION OF CASES REPORTED BY TROELL, JUNET AND MOORE

	Sex	Age	Acromegaly	Cranial Hyperostosis (entity and type)	Toxic Goiter or Simple Thyrotoxicosis	Diabetes Mellitus	Findings at Operation or Autopsy	Other Data
Troell[18]	F	47	+	+ (hyperostosis frontalis interna)	+		Chromophobe adenoma	
	F	41		+ (hyperostosis frontalis interna)	+			
Junet[11]	F	73	+	+ (hyperostosis frontalis + interna)	+		Eosinophilic adenoma	
	F	60	+	—	+	+	Eosinophilic adenoma	
	F	52	+	+ (hyperostosis calvariae + diffusa) +	+		Eosinophilic adenoma	Asthenia, headache
Moore[14,15]	F	42	+	+ (hyperostosis calvariae diffusa)	+ Basal metabolism 33% plus	+(?		
	F		+	+ (hyperostosis calvariae + diffusa)	+ Basal metabolism 32% plus	+	Eosinophilic adenoma	
	F	47	+	+ (hyperostosis calvariae + diffusa)	+ Basal metabolism 26% plus	+(?		Obesity, involutional psychosis
	F	23	+	+ (hyperostosis calvariae + diffusa) +	+	+	Eosinophilic adenoma	Headache
	F	51	+	+ (hyperostosis frontalis interna + hyperostosis calvariae diffusa)	+	+		Obesity
	F	41	+	+ (no findings + hyperostosis calvariae diffusa)	+ Basal metabolism 40% plus	+		

the process is quite localized; and (c) the difference in sex distribution of the 2 diseases.

Henschen,[8,9] too, maintains that calvarial changes found in acromegaly differ somewhat from typical hyperostosis frontalis interna, both morphologically and in the topographic distribution.

Among the more recent studies on skeletal alterations caused by acromegaly is the one by Juliani and Mauri.[10] In a group of 24 patients (16 women and 8 men) with ages varying between 26 and 61 years they found hyperostosis of the internal frontal table in 10 cases, diffuse thickening of the diploë and hyperostosis of the internal and external tables in 6 cases, and in 2 patients a typical picture of hyperostosis frontalis interna. The association of acromegaly and hyperostosis frontalis interna or hyperostosis

Table II

INCIDENCE OF ACROMEGALY ASSOCIATED WITH CRANIAL HYPEROSTOSIS AS REPORTED BY MOORE

Sex	Acromegaly	Acromegaloid Condition	Acromegaly + Hyperostosis	Acromegaloid Condition + Hyperostosis
Male	24	3	1	0
Female	18	3	5	3
Total	42	6	6	3

Note: The ages of Moore's patients ranged from 17 to 55 years in males and 18 to 53 years in females.

calvariae diffusa has also been described by Ellegast,[7] who recently reported a characteristic case.

The coexistence of thyroid changes with acromegalic disease has been noted rather frequently. Cushing[4] reported a case of acromegaly with a voluminous substernal goiter; Atkinson,[2] in an analysis of reported autopsied cases of acromegaly, found that only 15 per cent had a normal thyroid; Davis[6] records that 50 per cent of 166 cases of acromegaly had thyroid enlargement; and Moore[15] found the combination of thyroid toxicity and acromegaly in 3 of his cases.

MATERIAL

Our observations are based on a study of a group of 27 acromegalic patients, 14 males and 13 females. In the males, the ages ranged from 27 to 65 years, with an average of 45 years; in the females, the ages ranged from 14 to 65 years, averaging 42 years.

The somatic changes characteristic of acromegaly were present in all these patients although they were more pronounced in some cases than others. There existed in several of the patients endocrine and sensorial disorders (ocular disturbances, violent headache), psychic asthenia, and depression as well as complaints typical of hypophyseal disease. Violent headaches and osteoarticular pains were more frequent in women. Along with menstrual disturbances, diffuse adiposity was observed in 3 cases.

Hyperthyroidism was present in 1 male and 1 female, a slight goiter in 1 woman (uptake of I^{131}, 66 per cent at 24 hours) and moderate hyperthyroidism in 2 other women. The glycemia levels in the cases examined appeared to be within normal limits.

Roentgenologically, besides the skull changes, which will be more fully described below, symmetric gigantism of the extremities and, in almost one-third of the cases, vertebral changes typical of acromegaly (platyspondylia and megaspondylia, Erdheim-type modification, kyphosis) were present.

SITE AND DISTRIBUTION

Hyperostosis of the skull is generally diffuse, sometimes with varying thickness in different areas. In the order of frequency and degree, the process affects the frontal bones, the occipital bone and the parietal bones. The process seems, less often, to be localized, isolated or predominantly in the frontal bone (Fig. 1*A*; 2*A*; and 3, *A* and *B*).

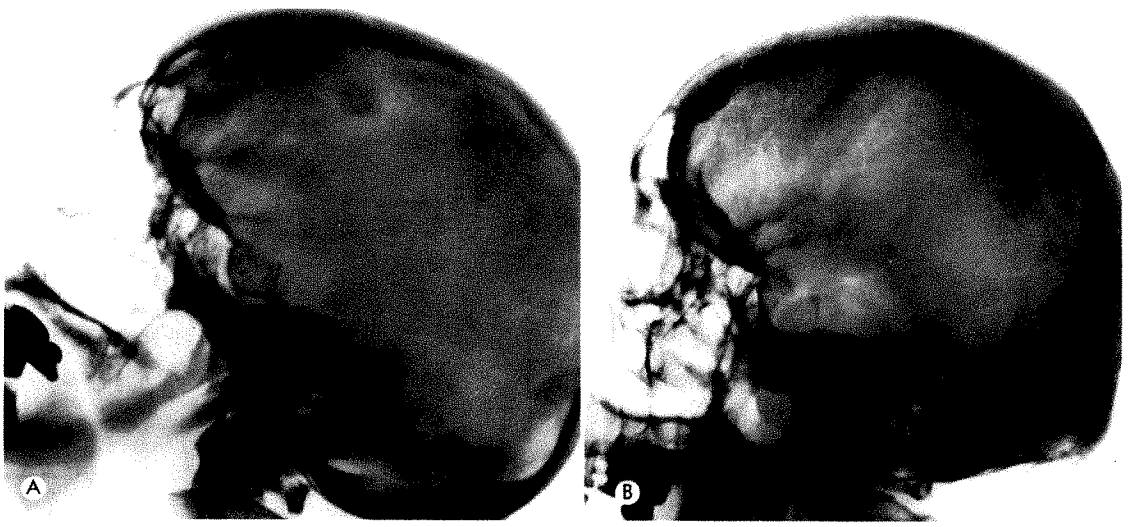


FIG. 1. Varying distribution and findings in cranial hyperostosis. (*A*) Male, aged 45 years, showing localized thickening of the internal table of the frontal bone. (*B*) Woman, aged 60 years, showing diffuse thickening of the cranial vault with areas of hyperostosis of the internal frontal table.

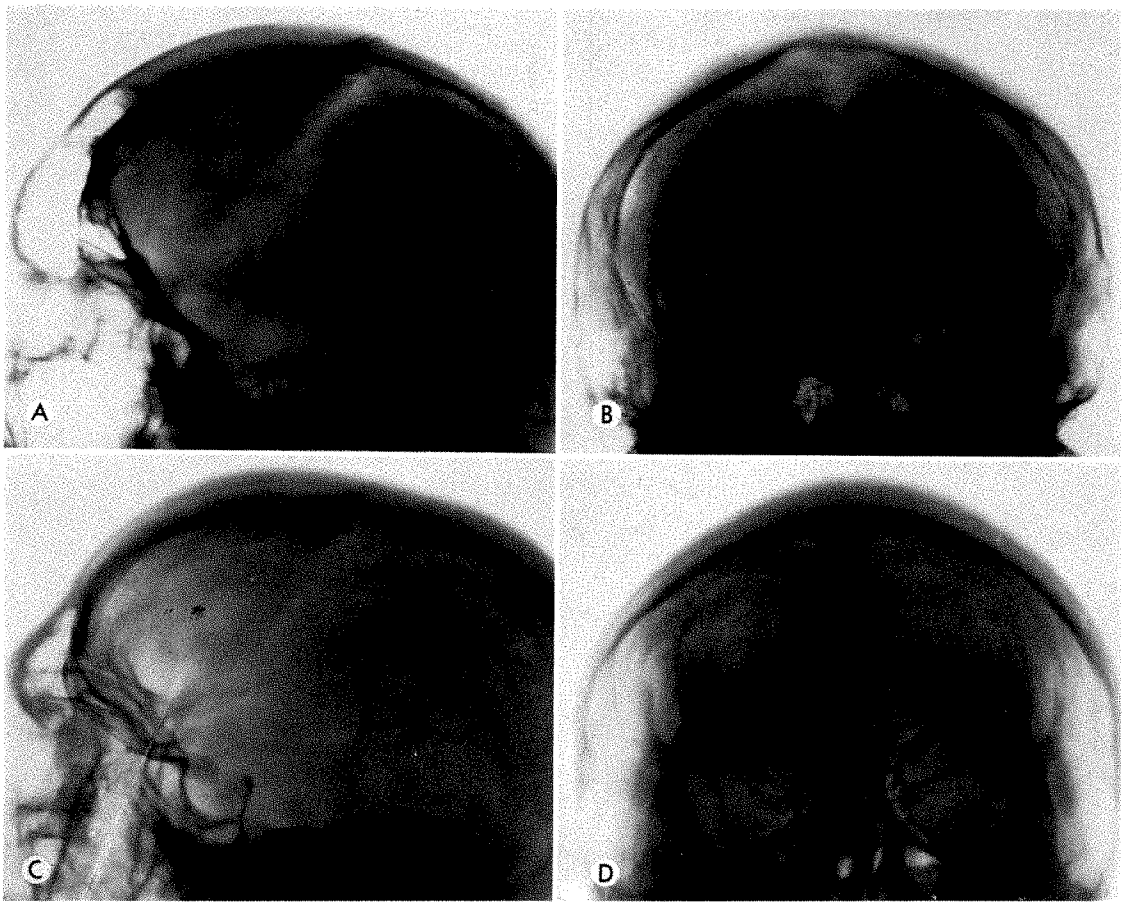


Fig. 2. Different aspects of hyperostosis of the cranial vault, in two right-angled projections. (*A*) Woman, aged 53 years, showing thickening of the frontal diploë with "scythe" appearance. (*B*) Same patient as in *A*. Anteroposterior roentgenogram showing considerable hyperpneumatization of the frontal sinuses, especially on the right. (*C*) Man, aged 58 years, showing diffuse thickening of the cranial diploë with initial signs of increased density of the internal table; no hyperpneumatization of the frontal sinuses is evident. (*D*) Same patient as in *C*. Anteroposterior roentgenogram showing that the diploë thickening is greatest at the top and decreases symmetrically on both sides.

The latter finding occurs in both sexes, though more frequently in women.

The greater incidence of hyperostosis of the frontal bone, at times affecting only the internal table and the diploë, and at other times occurring diffusely over the external table, is quite suggestive not only of Morgagni's syndrome, but also of certain cases of Paget's disease, fibrous osteitis and Cooley's disease. The pathogenetic reasons for this zonal electivity might be because of possible difference in vascularization in the bones of the skull.

In ascertaining the extent of hyperostosis, use of roentgenograms in both right angle projections, sometimes completed by tangential studies, is necessary. Superimposition of images, a difference in skull curvature, and the combination of various projective effects may sometimes give a false impression. Accurate evaluation of hyperostosis can be deduced only by combined visualization. For a detailed examination of the structures of the various parts of the vault of the skull, plesioroentgenography may also be of value.

In the present series, skull hyperostosis was found in 10 of the 14 males (1 case with frontal hyperostosis, 5 with diffuse hyperostosis, 4 cases with a combination of diffuse

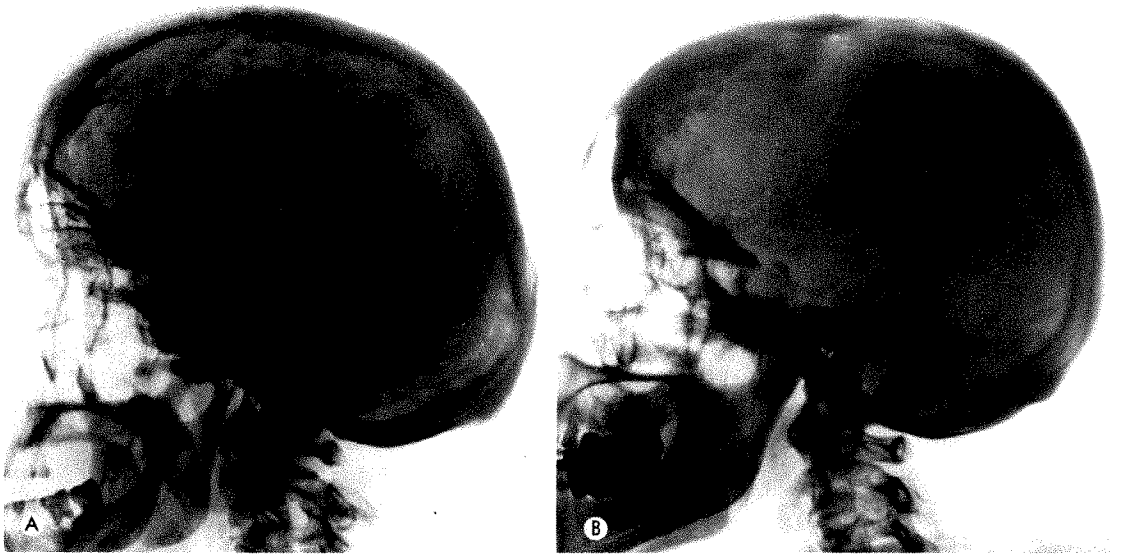


FIG. 3. Internal frontal hyperostosis in acromegaly. (*A*) Man, aged 53 years, showing widespread thickening of the cranial vault of moderate degree with evident increased density of the frontal bone. (*B*) Woman, aged 50 years, showing the typical Morgagni syndrome combined with acromegaly as well as internal frontal hyperostosis of the tuberous type.

and frontal hyperostosis) and in 11 of the 13 females (6 with frontal hyperostosis, 5 diffuse and 1 combined).

RELATIONSHIP BETWEEN THE EXTENT OF HYPEROSTOSIS OF THE SKULL AND DEVELOPMENT OF OTHER CRANIAL BONES AND BETWEEN FRONTAL HYPEROSTOSIS AND DEVELOPMENT OF FRONTAL SINUSES

From a comparative analysis of the roentgenograms of the skull of our patients, we tried to determine whether a relationship exists between the hyperostotic process of the vault and the development of the facial bones and frontal sinuses. There appears to be a certain proportional ratio between the development of the facial bones and the extent of skull thickening, especially in diffused hyperostosis and in males (Fig. 5). This ratio, however, is not necessarily constant or characteristic since, although patients with thickened and dense skulls generally showed considerable development of the facial bone, marked facial development was also noted in patients with osteoporotic skulls of average thickness (Fig. 6, *A* and *B*).

The degree of pneumatization of the frontal sinuses generally was not found to be proportional to the frontal hyperostotic process. In some cases, however, in which there was a uniform thickening of the diploë, limited to or predominant in the frontal bone, with moderate osteoporosis in large patches and without tuberosity or thickening of the internal table, an abnormal development of the frontal sinuses was noted (Fig. 2, *A* and *B*). One might suppose from these findings that there may exist a causal link between the two phenomena in that the hypertrophy of the diploic tissue with thinning of the osseous trabeculae may stimulate pneumatization of the frontal sinuses, probably due to osteoclastic and reabsorption activity. This is also suggested from observations of the abnormal development of the frontal sinuses in certain cases of Paget's disease (Fig. 7) and in some cases of polyostotic fibrous dysplasia.[3,17]

MORPHOLOGIC FEATURES OF HYPEROSTOSIS OF THE SKULL

The structural aspect of the hyperostotic skull may vary to a certain extent. This is determined by the variation in the thicken-



Fig. 4. Internal frontal hyperostosis of the tuberous type associated with acromegaly. (*A* and *B*) Man, aged 47 years. Roentgenographic aspect in the two right-angled projections.

ing process of the diploë, and whether or not the disease is associated with osteoporosis, or related to the uniformity of the thickening of the internal table.

Hypertrophy of the diploë may extend to the whole skull, either in a uniform or non-uniform manner. The trabeculae of the spongy bone often appear to be thin, especially in older patients, with reduction of the calcium content. On left lateral roentgenograms the vault may show a "scythe" appearance caused by gross thickening in the frontal bone (Fig. 2*A*); in some cases the calvaria is rather large with a markedly developed occipital protuberance (Fig. 4, *A* and *B*). Anteroposterior roentgenograms demonstrate the thickness of the skull as being greatest at the top and decreasing (a) symmetrically and uniformly towards the parietotemporal region or, (b) asymmetrically on the two sides or, (c) limited almost exclusively to the top and rapidly decreasing on the sides (Fig. 2, *A* and *B*; 4*B*; and 6*B*).

With thickening of the diploë, it is not uncommon to find true osteosclerosis with an eburneous appearance. The coexistence of more or less uniform thickening of the internal table contributes to better roentgenographic demonstration of the massiveness of the skull, sometimes even masking the distribution of the vascular grooves and diploic channels.

Roentgenographically, the bone thickening appears more evident in those cases in which hyperostosis is localized to the internal frontal table, giving an irregular tuberous aspect typical of Morgagni's syndrome (Fig. 4, *A* and *B*). There may be other com-

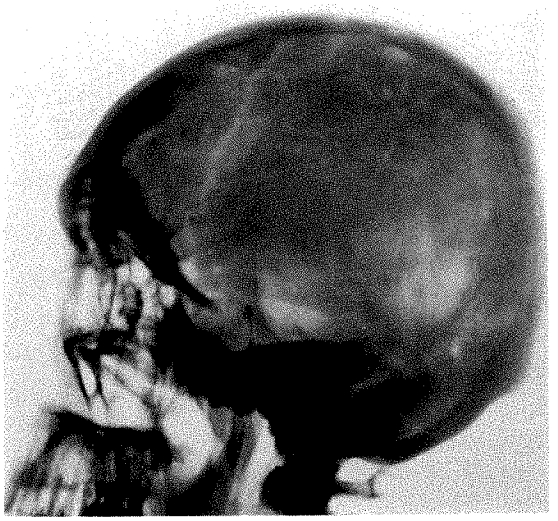


Fig. 5. Typical cranial acromegaly with hyperostosis. Man, aged 58 years, showing considerable thickening of the frontal bone, while the frontal sinuses appear almost hypoplastic.

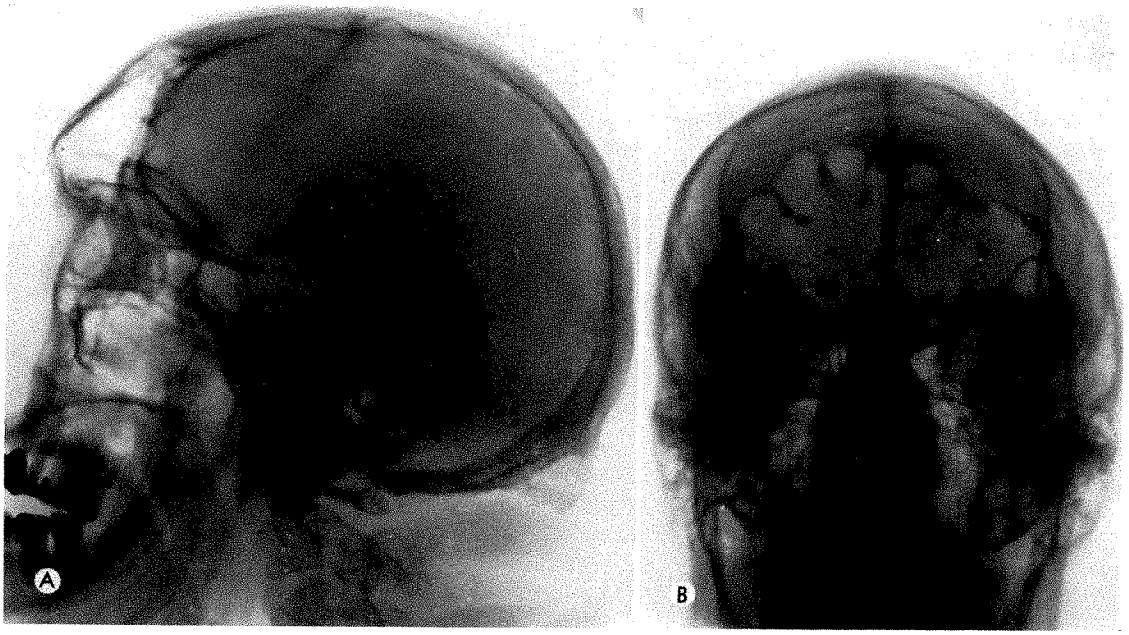


FIG. 6. Acromegalic cranium without hyperostosis. (*A*) Man, aged 50 years, with osteoporosis of the cranial vault showing well-developed external occipital protuberance and prognathism of the facial bones. (*B*) Same patient as in *A*. Anteroposterior roentgenogram showing a rather thin cranial vault and marked hyperpneumatization of the frontal sinuses.

bined patterns, such as diffuse diploic thickening associated with zones of "plaque" or "cloud" hyperostosis predominantly in the internal frontal table and, to a lesser degree, in the occipital and parietal bones.

CONCLUSIONS

From an analysis of 27 cases of acromegaly, the following conclusions were derived:

1. Thickening of the skull is found in a rather high percentage of cases in both sexes.

2. Most often, hyperostosis affects the diploë, which on roentgenograms appears thickened. In a certain number of cases, diminution of the calcium content is noted, sometimes with evident diploic grooves. Fairly frequently, roentgenograms show, instead, a more or less diffuse osteosclerosis with increase in density also of the internal table.

3. Thickening may occur throughout the skull either symmetrically or in varying segments; the frontal bone is most often affected.

4. Besides cases in which diffuse thickening and increase in the density of the skull are seen, there are, more rarely, cases showing internal frontal hyperostosis with features practically analogous to the typical picture of Morgagni's syndrome. This, though found in both sexes, is more frequent in women.

5. It seems that a certain relationship exists, although not necessarily characteris-



FIG. 7. Hyperpneumatization of the frontal sinuses associated with considerable thickening of the skull, especially of the frontal bone, in Paget's disease. Woman, aged 54 years.

tic of the disease, between the development of the facial bones and hyperostosis of the skull. This relationship does not appear to be proportional to the degree of development of the frontal sinuses.

The association of thyroid disorder and diabetes (so-called "Troell-Junet syndrome," proposed by Moore) was too infrequently encountered in our cases to be of any significant value. In fact, hyperthyroidism was present in only 3 women, while in 2 other cases there was hypothyroidism; no cases of diabetes were noted. A Morgagni-type picture concomitant with acromegaly was noted in 3 women.

From our material we have not been able to detect a constant relationship between the appearance of hyperostosis and the onset of clinical symptomatology in acromegalic disease. In some cases which we were able to follow roentgenographically for 10 years and more, we have noted that the hyperostotic process was practically stationary rather than progressive in character.

Of interest also is the fact that the clinical symptomatology, in particular visual disturbances and severe headaches, was not proportional to the degree of hyperostosis of the vault of the skull.

SUMMARY

An analysis of the morphologic roentgen features of hyperostosis of the skull was made in acromegaly.

In a group of 27 patients (14 males and 13 females), the structure of the skull, the type of disease, and the degree and zonal distribution of the hyperostotic process were considered in an effort to evaluate the frequency and proportional relationship of skull thickening to the development of the facial bones.

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THE SMALL SELLA TURCICA*

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THE present study is an attempt to evaluate the diagnostic roentgenographic significance of the "small sella turcica."

PREVIOUS ROENTGENOGRAPHIC OBSERVATIONS

The previous reports dealing with the "small sella turcica" are summarized in Table 1. In all of these investigations, the size of the sella turcica was estimated from evaluations of its lateral roentgenographic profile. Since the third dimension (width) of the sella is quite variable, however, the sella size cannot be reliably estimated from the lateral roentgenograms alone.[25,26] In the present study, the "small sella turcica" was studied volumetrically using the formula: $V=\frac{1}{2}(W\times H\times L)$ where V is volume, W width, H height and L length.

THE PSEUDO-SMALL SELLA TURCICA

The importance of evaluating the sella turcica three-dimensionally is dramatically illustrated by our study of myotonic dystrophy. In agreement with previous reports,[20,47,78,84,90] we frequently found a small sella turcica on the lateral skull roentgenogram in a group of 39 adult patients affected with this muscle disorder. Even using strict criteria (length 8 mm. or less; height 6 mm. or less), the sella turcica was small on lateral profile in 6 cases. When the size of these sellas was evaluated volumetrically, however, it was found to be normal in every case due to their predominantly large widths (Fig. 1, *A* and *B*). Postmortem studies also indicate that the pituitary gland is normal in myotonic dystrophy.[12,18,20,78,92] One patient in our series at autopsy showed a normal 0.6 gm. pituitary gland.

TABLE I

PREVIOUS ROENTGENOGRAPHIC OBSERVATIONS OF THE "SMALL SELLA TURCICA"

Authors	Small Sellas Observed	Associated Conditions
Cushing[34] (1912)	Some	Hypopituitarism in children
Biedl[10] (1922)	Some	Hypopituitary dwarfism
Haas[39] (1925)	7	Hypo- and hyperthyroidism, hypoparathyroidism, precocious puberty
Fründ[31] (1927)	5	Hypopituitarism
Reinert[71] (1927)	15	Infantilism, adiposogenital dystrophy, hypothyroidism, trauma, asthma
Erdelyi and Scholtz[28] (1928)	38	Endocrine disturbances in 24 cases
Steiert[86] (1928)	37	Lipodystrophy, progressive muscular dystrophy, proportioned dwarfism
Gordon and Bell[34] (1936)	Some	Various unrelated disorders
Martin[59] (1940)	161	Endocrine and psychiatric disorders
Carstens[19] (1949)	24	Various unrelated disorders
Jungmayr[47] (1951)	14	Myotonic dystrophy
Lohmann[57] (1951)	208	Various psychiatric, neurologic and endocrine disorders
Caughey[20] (1952)	13	Myotonic dystrophy
Walton and Warrick[90] (1954)	17	Myotonic dystrophy
Ott[66] (1955)	100	Various unrelated disorders
Kovacs and Goth[51] (1958)	36	Pituitary hypofunction in all cases

* From the Section on Neuroradiology (Head, Giovanni Di Chiro, M.D.), National Institute of Neurological Diseases and Blindness, National Institutes of Health, Bethesda, Maryland.

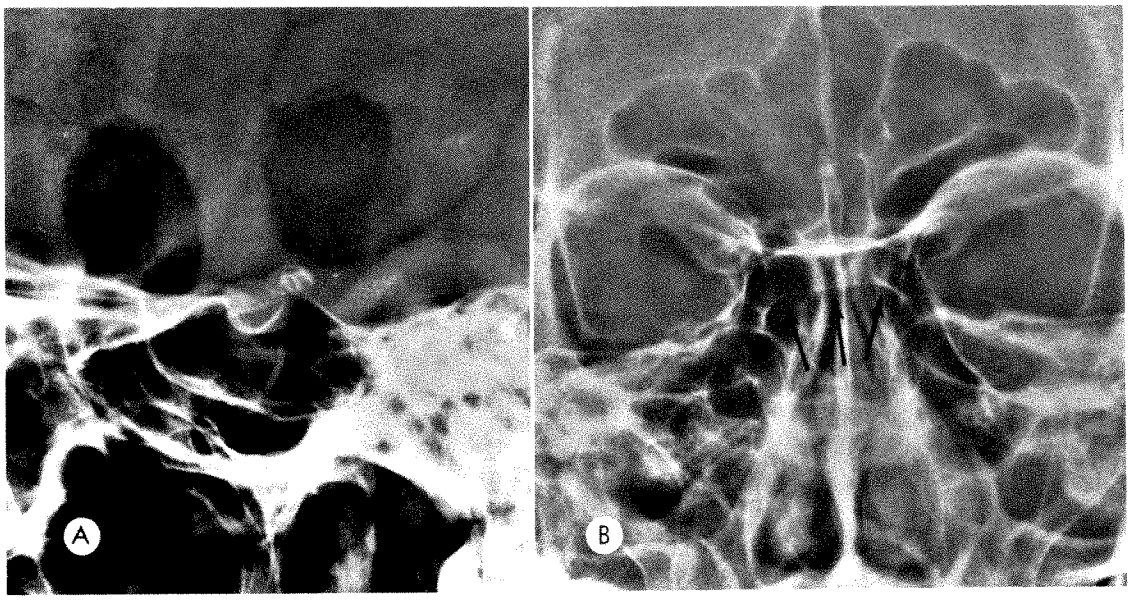


Fig. 1. (*A*) Pseudo-small sella turcica in a case of myotonic dystrophy; because of unusually large width (*B*) volume is normal. (Burr holes for previous subdural hygroma.)

We have termed the sellas which appear small on lateral skull roentgenograms but which have large widths, "pseudo-small," as they present a false impression of the sella and pituitary size. A further example of a pseudo-small sella turcica which contained an unusually wide pituitary gland may be seen in Figure 2, *A* and *B*.

THE SMALL SELLA TURCICA

The normal range for sella volume in adults has been previously investigated roentgenographically.[26] To enable us to study the sella turcica during its period of growth, we extended these volumetric determinations to 347 individuals ranging from birth to 26 years of age. This "normal" control population consisted largely of cases of head trauma, neuromuscular disease (excluding myotonic dystrophy), acute leukemia and cases of idiopathic and post-traumatic epilepsy showing normal pneumoencephalograms.

The results of this study are presented in Figure 3. A considerable variation of the sella size in each age group and a rapid increase of the volume of the sella during the first 3 years followed by a gradual growth

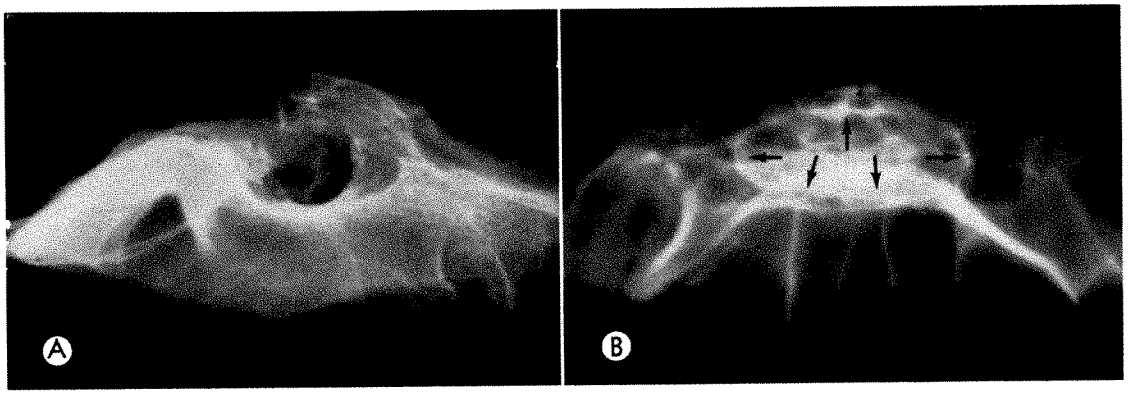


Fig. 2. Sella specimen with small lateral profile (*A*) and large width (*B*). Arrows in *B* outline correspondingly wide barium-coated pituitary gland.

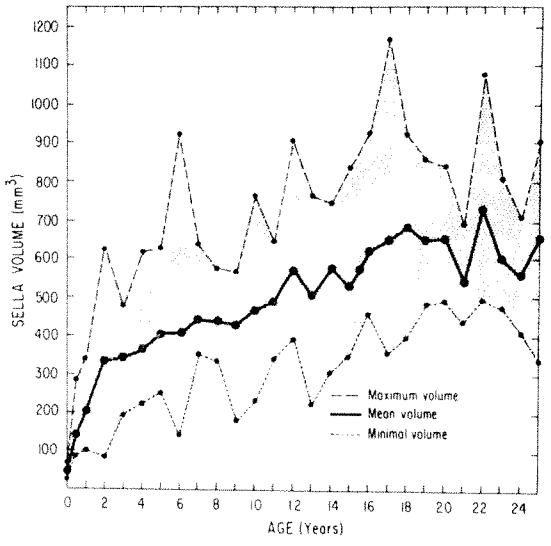


FIG. 3. Sellar volumes of 347 "normal" controls.

to 18 years are observed. Based on our experience, the minimal sella volumes to be expected in normal individuals are shown in Figure 4. Lower values are considered indicative of small sellas, particularly in the older age groups. In the young and the very young (below 3 years), the lower values are less significant due to the great developmental variation.

To establish the validity of the volumetric estimation of sella size in growing children, the previous anatomo-roentgenographic comparison of Di Chiro and Nelson[26] was extended to include 22 fresh cadavers of individuals ranging in age from 3 to 16 years. The relationship between the roentgenographic volume of the sella and the pituitary volume and observed

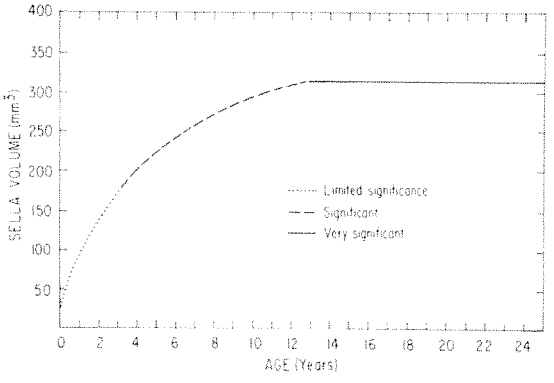


FIG. 4. Minimal expected normal sella volumes.

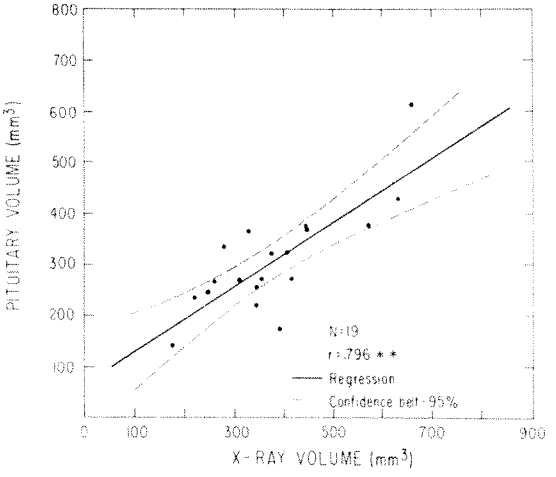


FIG. 5. Relationship between roentgenographic volume of the sella turcica and pituitary volume in 19 cases shown by the equation: Pituitary volume = .627 roentgen-ray volume + 66.232.

volume of the sella cast is represented in Figures 5 and 6, respectively. The average error in the estimation of the sella cast size and pituitary volume was 27.1 per cent and 20.8 per cent respectively, which is probably satisfactory for clinical purposes.

THE SMALL SELLA TURCICA IN THE GENERAL HOSPITAL POPULATION

Having defined the small sella turcica, an

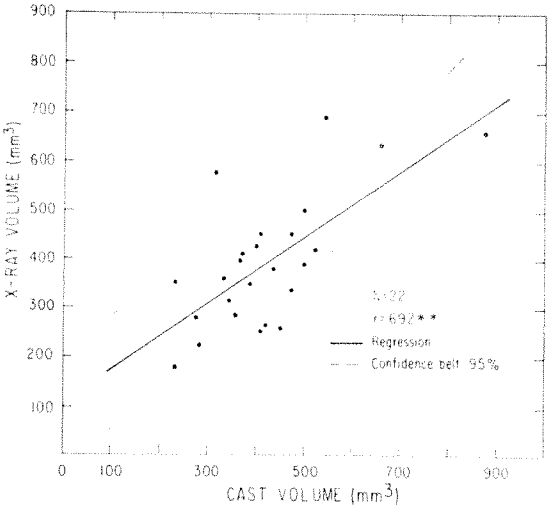


FIG. 6. Relationship between roentgenographic volume of the sella turcica and observed volume of the sellar cast in 22 cases shown by the equation: roentgen-ray volume = .681 cast volume + 103.342.

attempt was made to determine its incidence and possible significance. Following the lead of previous investigators,[19,57] random hospital cases were studied. The plain skull roentgenograms of 500 general hospital patients and 500 mental patients ranging in age from 2 to 99 years were examined. There was a predominance of elderly and middle-aged individuals but the sex distribution was approximately equal. The study was focused on the lateral skull roentgenogram, as the volumes were calculated (except in children) only in sellas having a height of 6 mm. or less or a length of 8 mm. or less on this view. Certain small sellas (having relatively normal height and length but exceptionally small width) were possibly overlooked. This procedure, however, provided a good method of checking previous two-dimensional studies.

In 36 adult patients, sellas having at least one small lateral dimension were discovered. In 7 of these, one of the lateral dimensions was relatively large. Therefore, small sella areas on the lateral roentgenograms were observed in about 3 per cent of the total cases in this series as in previous two-dimensional studies.[19,57] However, when the width was considered, the total volume was within normal limits in all but one case. This sella, with a volume of 158 $mm.^3$, was present in a 5 year old girl examined after head trauma. Thus, in this random study, the small sellas encountered were all pseudo-small with one exception. It was then evident that the attention had to be focused on specific conditions in which a small sella turcica might be theoretically expected.

THE SMALL SELLA TURCICA IN HYPOPITUITARISM

Since a correlation between sella size and pituitary size has been noted in the anatomic studies of many previous investigators,[26] it seemed reasonable to investigate conditions associated with small pituitary glands. As the sella turcica has never been shown to reduce in size, except in recovery from pathologic erosions, we concentrated our attention further on the pituitary gland at birth and during the period of sella development.

Congenital absence of the pituitary gland has been reported by several authors.[13,16,70] The sella size was noted, however, only in the case of Blizzard and Alberts[13] where it was definitely small. In anencephaly, which is usually associated with a hypoplastic or dystopic pituitary gland,[1,13,22] an absent or poorly formed sella turcica is a well-known finding.[1,22] The intrasellar pituitary gland has also been reported small in 18 of 21 cases of nontumoral, hypopituitary dwarfism (Table II). Hypopituitary dwarfism seemed, therefore, suitable for a clinical, roentgenographic investigation of sella turcica size with particular regard to the small sella.

Forty-four patients ranging in age from 1 to 49 years were selected for study. The diagnosis of idiopathic hypopituitary dwarfism was considered established (Table III) when the patients showed marked retardation of height age, bone age, infantilism and one or more of the following: (1) a marked clinical response to injected human growth hormone; (2) an insufficiency of adrenocorticotrophic hormone, indicated by a lack of response to SU-4885, an abnormal water load test or an abnormal insulin tolerance; (3) an insufficiency of thyrotrophic hormone, indicated by abnormally low thyroid activity, increased by injecting T.S.H.; and (4) low or absent gonadotrophins in patients over the age of 20 years. Almost all of the patients had a history of normal birth weight and a normal development in early infancy. Patients with intracranial tumors or with onset of hypopituitary symptoms after the age of 6 years were excluded.

Twenty-five hypopituitary patients (56.8 per cent) had small sellas; only 6 patients (13.6 per cent) had sellas of average size, while the remainder had low normal volumes (Table III). In 3 additional cases of hypopituitary dwarfism of late onset (ages 7–11 years) the sellas were of normal size. While in most cases the sella turcica was

merely small (Fig. 7, *A* and *B*), in about one-third of the cases, we noted a shallow shape of the lateral profile (Fig. 8, *A* and *B*), previously observed by others.[28]

In an attempt to evaluate whether the small sellas in the hypopituitary dwarfs were a reflection of the pituitary size or merely of the small general body size, sellas were investigated in other conditions associated with dwarfism—achondroplasia, gonadal dysgenesis, hypothyroidism, congenital or genetic dwarfism, and dwarfism secondary to nutritional, central nervous system, cardiac and renal disease. The

TABLE II

POSTMORTEM PITUITARY PATHOLOGY IN NONTUMORAL CASES OF HYPOPITUITARY DWARFISM

Authors	Age and Sex	Height (cm.)	Pituitary Gland	
			Weight or Dimensions	Gross Appearance
Hueter[44] (1905)	42 F	106		Small
Kraus[53] (1918)	27 F	121	0.16 gm.	Hypoplastic
Simmonds[83] (1919)	21 M	110	0.2 gm.	Extremely small; large anterior lobe cyst
Berblinger[9] (1920)	19 M	144	0.8 gm.	Anterior lobe large
Sternberg[86] (1920) Case 1	17 M	92		Very thin and flat
Sternberg[86] (1920) Case 2	20 M	126		Normal
Priesel[68] (1920)	91 M	132		Dystopy of posterior lobe; small cystic anterior lobe in sella
Schultz[77] (1924)	18 M	145	12.5×5×11 mm.	Atrophy of anterior lobe
Koch[50] (1926)	11 F	94	0.7 gm.	Large cyst of anterior lobe
Sternberg[87] (1929)	18 M	135	0.4 gm.	Small
Kraus[56] (1932)	26 M	138	0.28 gm.	Dystopy of posterior lobe
Katzenstein[49] (1933)	51 F	131		Small
Montanini[61] (1934)	67 F	123	0.25 gm.	Small
Biemond and Hartz[11] (1935)	31 F	150	0.3 gm.	Small; dystopic posterior lobe
Ceranke[21] (1936)	50 F	129		Extremely small
Farina[30] (1942)	62 F	126		Hypoplastic
Grünthal and Keller[37] (1943)	26 M	122		Posterior lobe absent; small anterior lobe
Hewer[43] (1944)	76 F	119	0.31 gm.	Small
Hedinger and Hürzeler[42] (1953)	21 M	145	0.2 gm.	Dystopy of posterior lobe; small anterior lobe
Ehrlich[27] (1957)	1 F	61		Empty sella; small dystopic gland at base of brain
Sandison[76] (1958)	15 F	120		Small

TABLE III

SIGNIFICANT DATA IN CASES OF HYPOPITUITARY DWARFISM

Case No.	Age and Sex	Height (cm.)	Human Growth Hormone Response	ACTH Function	TSH Function	Gonadotrophic Function	Sella Size
1	10 M	102	+	A	Q		Small
2	49 M	151		A	N	A	Small
3	15 M	146		A	N		Normal (borderline small)
4	6 F	90	+	A	N		Small
5	18 F	145		A	Q		Small
6	12 M	130		A	Q		Normal
7	11 M	110	+	Q	Q		Small
8	8 M	107		Q	A		Normal (borderline small)
9	4 M	69	+	A	Q		Small
10	1½ F	53		A	A		Small
11	20 M	142		A	N		Normal (borderline small)
12	17 M	140		A	A		Normal (borderline small)
13	36 M	152		N	N	A	Small
14	8 M	99		N	A		Small
15	15 F	123	+	A	A		Normal (borderline small)
16	8 M	102		N	A		Small
17	14 M	126		A	A		Small
18	8 F	94	+	Q	N		Small
19	7 M	89		Q	A		Normal (borderline small)
20	8 M	102		N	A		Small
21	7½ F	90	+	A	A		Small
22	12 M	123		A	A		Normal (borderline small)
23	5 M	87		Q	A		Small
24	14 F	135		A	Q		Normal (borderline small)
25	44 F	122		N	N	A	Normal
26	8 M	110	+	A	N		Normal
27	10 F	115		A	N		Normal
28	8½ M	109		A	A		Small
29	10 M	116		A	Q		Small
30	8½ M	97		A	Q		Small
31	10 M	109		A	Q		Normal (borderline small)
32	16 M	136		A	A		Small
33	11 M	107	+	N	N		Normal (borderline small)
34	14½ F	124	+	A	Q		Small
35	8 M	106		A	Q		Normal
36	3 F	68		A	Q		Normal (borderline small)
37	14 M	120		A	N		Normal (borderline small)
38	13 M	121		A	N		Small
39	17 M	142		A	Q		Small
40	16 F	141		A	N		Normal
41	8 M	104		A	N		Small
42	6 M	99		A	N		Normal (borderline small)
43	17 F	135		A	A		Small
44	9 F	116		N	A		Small

Key: A = abnormal; Q = questionably abnormal; N = normal.

pituitary gland has been found to be of normal size in gonadal dysgenesis[2,29,36,63,64,67,69,78,80,89] and achondroplasia.[38,52,58,62,66,81] Enlargement of the pituitary gland has been noted in hypothyroidism.[6,8,15,17,74] Two autopsy reports in "bird-headed" dwarfs[79] and one in a well proportioned nonhypopituitary dwarf[75] suggest that the pituitary

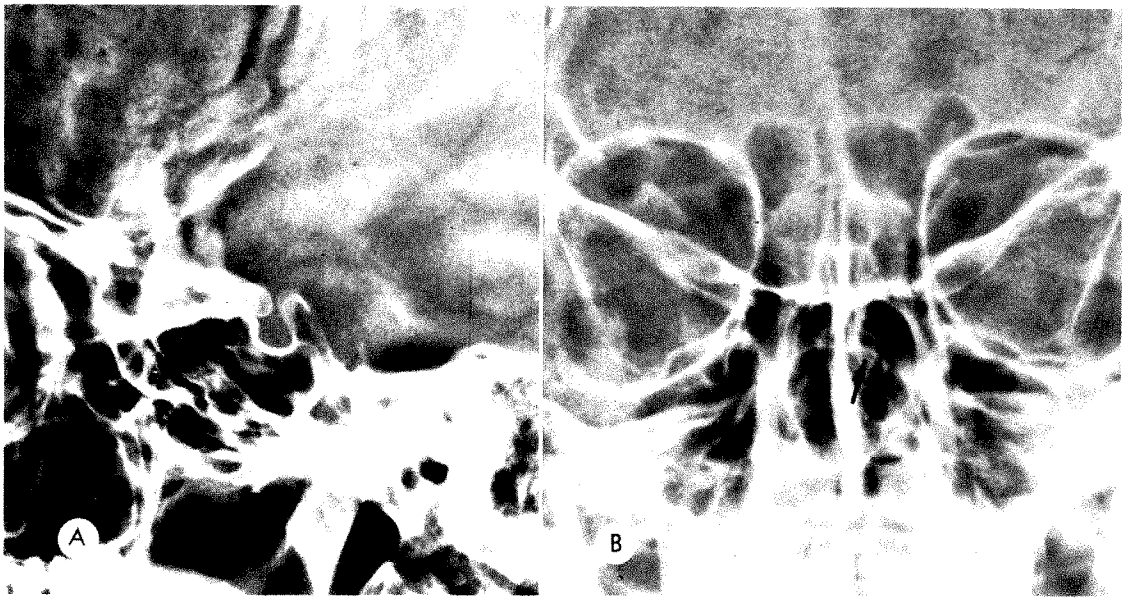


FIG. 7. (*A* and *B*) Small sella of 17 year old hypopituitary dwarf.
(Courtesy of Drs. Judson J. Van Wyk and Francis D. Pepper.)

is somewhat small in genetic dwarfism. Normal pituitaries have been described in renal dwarfism.[3] Information pertaining to the pituitary is lacking in dwarfism secondary to malnutrition and other chronic illnesses.

The skull roentgenograms of 14 patients with achondroplasia, 18 with gonadal dysgenesis, 24 with hypothyroidism and 15 with dwarfism associated with chronic illnesses were studied. Seventy-four patients, in whom no specific etiology could be established, were investigated as genetic dwarfs. Small sellas were found in 10 (13.5 per cent)

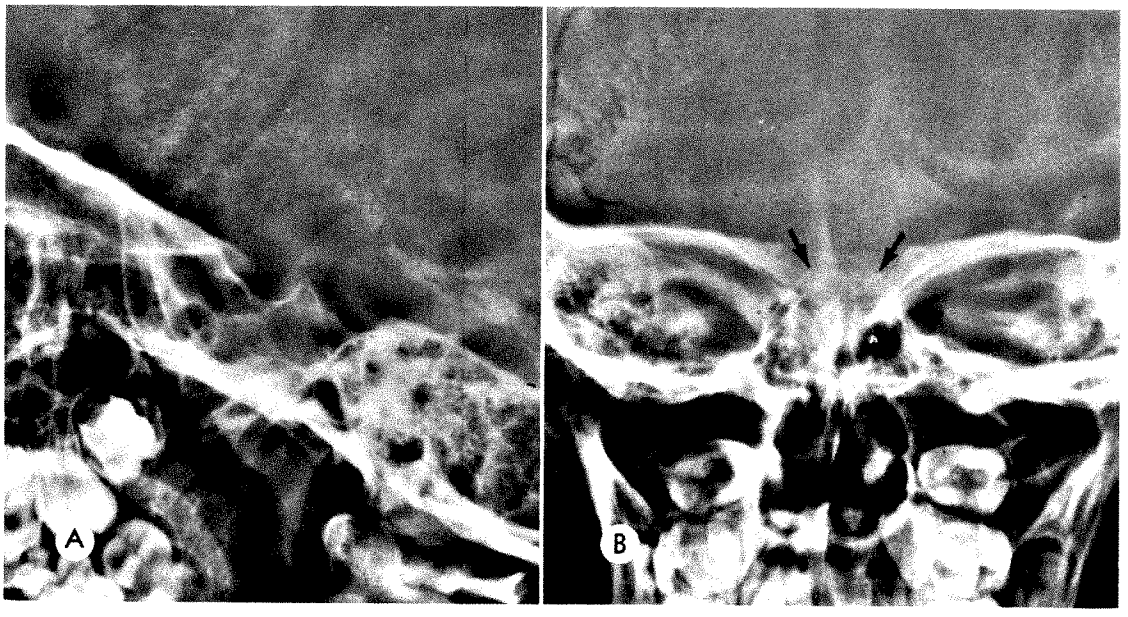


FIG. 8. (*A* and *B*) Small sella of 8 year old hypopituitary dwarf. Note shallow profile in *A*.
(Courtesy of Drs. Judson J. Van Wyk and Francis D. Pepper.)

TABLE IV

MEAN SELLA TURCICA VOLUMES IN CONTROL AND DWARF PATIENTS OF EQUIVALENT BODY HEIGHTS

Patient Heights (cm.)	Mean Sella Volumes (cubic mm.)						
	Control Patients	Genetic Dwarfs	Achondroplastic Dwarfs	Hypothyroid Dwarfs	Gonadal Dysgenesis Patients	Hypopituitary Dwarfs	Dwarfs with Chronic Illness
50–59	39 (6)*	78 (1)	— —	98 (3)	— —	58 (1)	— —
60–69	143 (10)	168 (8)	— —	228 (2)	169 (2)	159 (2)	— —
70–79	202 (12)	189 (5)	358 (2)	308 (5)	220 (2)	— —	— —
80–89	333 (16)	295 (7)	326 (2)	390 (3)	195 (1)	216 (2)	251 (6)
90–99	344 (22)	296 (12)	438 (2)	455 (1)	— —	190 (5)	222 (2)
100–109	382 (38)	292 (5)	— —	496 (1)	— —	268 (9)	369 (4)
110–119	432 (31)	386 (20)	654 (1)	565 (2)	416 (1)	331 (5)	— —
120–129	442 (16)	311 (2)	619 (1)	978 (2)	553 (2)	340 (7)	485 (2)
130–139	448 (26)	604 (4)	— —	672 (2)	675 (1)	317 (4)	— —
140–149	522 (24)	401 (3)	— —	— —	600 (2)	379 (6)	— —
150–159	549 (28)	540 (1)	— —	720 (1)	540 (2)	196 (2)	— —
160–169	650 (36)	— —	— —	— —	614 (2)	— —	— —
170–179	630 (72)	— —	— —	— —	— —	— —	— —

* Figures in parentheses indicate the number of patients considered in the averaged volumes.

genetic dwarfs and 2 (13.3 per cent) cases of dwarfism associated with chronic illness. No small sellas were found in dwarfism associated with achondroplasia, hypothyroidism or gonadal dysgenesis.

The mean sella turcica volumes of dwarfs and controls of equal height were then compared (Table IV). The mean sella turcica volumes of the hypopituitary patients were smaller than all of the other groups considered. The sella turcica volumes of the achondroplastic and gonadal dysgenesis patients were similar to those of the controls. As previously noted by others,[4,82,88] the hypothyroid patients had consistently larger sellas than the controls (Fig. 9, *A* and *B*). The sella turcica volumes of patients with genetic dwarfism and dwarfism associated with chronic disease lay generally between those of the control and hypopituitary patients. Thus, there was a distinct correlation between the sella turcica volume and the size of the pituitary gland inferred in each group from the pathologic literature.

The correlation between pituitary pathology and sella turcica volume was then evaluated in other conditions unassociated with dwarfism but characterized by deficient pituitary function—idiopathic diabetes insipidus and hypogonadotrophic hypogonadism. Mongolism, in which a pituitary disorder has long been speculated,[5] was also included in the investigation. In diabetes insipidus, the pituitary glands have been either normal[14,41] or small due to atrophy of the posterior lobe.[14,33,55] Well documented pathologic studies of the pituitary gland in idiopathic hypogonadotrophic hypogonadism unassociated with dwarfism have not been reported,[46] although pituitary glands of variable gross appearance have been noted in nontumoral adiposogenital dystrophy,[35,72] infantilism,[45] and eunuchoidism of undetermined etiology.[32] Pituitary hypoplasia has been reported in mongolism[5,6] though the gland is usually of average size.[7]

Nine patients with idiopathic diabetes insipidus, 6 patients with idiopathic hypogonadotrophic hypogonadism, and 65 cases of mongolism were investigated. The sella turcica size was normal in diabetes insipidus and hypogonadism except for a small sella turcica in one case of diabetes insipidus, in which dwarfism and suspected anterior pituitary hypofunction were also present. In mongolism, the sella turcica

size was normal in all but 5 cases (7.7 per cent) which had small sellas.

The sella turcica size was finally investigated in other childhood endocrine disorders—hypoparathyroidism, hyperthyroidism, primary hypogonadism, Cushing's syndrome and Addison's disease, many of which have previously been implicated with small sellas.[19,39,59] However, we have not found reports of small pituitary glands in these conditions[8,23,46,54] with the exception of hyperthyroidism.[54] No small sellas were found in 7 cases of hypoparathyroidism (idiopathic and pseudohypoparathyroidism), 9 of hyperthyroidism, 13 of Cushing's syndrome, 5 of primary hypogonadism and 4 of Addison's disease.

DISCUSSION

The small sella turcica is an extremely rare finding in the average hospital population and does not exist in adults except in dwarfs. Small sellas, on the other hand, are found in the majority of patients with hypopituitary dwarfism. Occasionally, small sellas are encountered in genetic dwarfism, mongolism and dwarfism due to chronic illness. The particularly high incidence of small sellas in hypopituitary dwarfism seems primarily related to the hypoplastic pituitary characteristic of this condition, rather than the small size of these patients. The investigations of others[20,48] seem to indicate a relatively insignificant relationship between the skull size and body height and the sella turcica size in normal subjects. In our own study, controls and nonhypopituitary dwarfs showed larger mean sella turcica volumes than hypopituitary patients of the same height. With the possible exception of 2 cases of dwarfism secondary to chronic illness, small sellas were found only in those conditions in which small pituitary glands have been reported. When the pituitary pathology occurred after the relatively complete development of the sella turcica—hypopituitary dwarfism with onset after the age of 6 years, diabetes insipidus, and possibly hypogonadotrophic hypogonadism—small sellas were not found. The normal sellas in hypopituitary dwarfism may be explained, at least in a certain percentage of cases, by intrasellar pituitary or subarachnoid cysts.

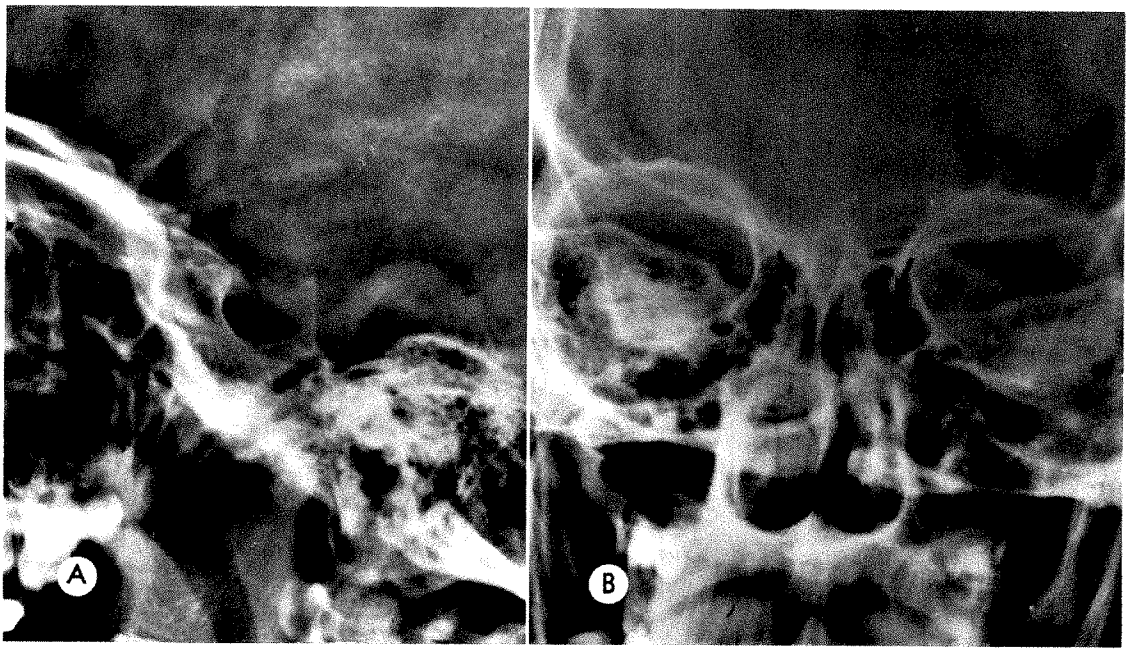


FIG. 9. (*A* and *B*) "Borderline large" sella of 13 year old hypothyroid dwarf.

The diagnosis of idiopathic hypopituitary dwarfism in prepubertal children is difficult and dependent to a large degree upon indirect chemical estimations of various trophic hormone deficiencies of the pituitary gland.[60,91] The finding of a sella turcica volume below the normal range for the patient's age should suggest and should be considered as confirmatory evidence of hypopituitarism in the doubtful cases. In patients with clinical and laboratory evidence of hypopituitarism, a small sella turcica by indicating primary pituitary hypoplasia should discourage further studies (*i.e.*, pneumoencephalography) to rule out sellar or suprasellar tumors.

SUMMARY

The previous studies on the small sella turcica are reviewed. The frequent finding of sellas with small lateral profiles but with unusually large widths emphasizes the necessity for a three-dimensional evaluation of sella size. The minimum normal volumetric values are defined for each given age, and values below these are considered indicative of a small sella turcica.

In the random hospital population, the small sella turcica is an extremely rare entity. In idiopathic hypopituitary dwarfism, however, the small sella turcica is observed in 56.8 per cent of the cases. This high incidence seems primarily related to hypoplasia of the pituitary gland rather than the general small body size of these patients. Small sellas are seen occasionally in mongolism, congenital dwarfism and dwarfism secondary to chronic illness, but in no other endocrine conditions. The small sella turcica suggests hypopituitarism and is of value in the differential diagnosis of hypopituitary dwarfism.

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THE ROENTGEN ANATOMY OF THE LATERAL WALLS OF THE ORBIT (ORBITAL LINE) AND THE MAXILLARY ANTRUM (ANTRAL LINE) IN THE SUBMENTOVERTICAL VIEW*

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THE lateral walls of the maxillary antrum and the bony orbit are identifiable on submentovertical roentgenograms of the skull. Both of these structures cast well-defined linear "bone" shadows on roentgenograms in close proximity to the curvilinear "bone" shadow of the great wing of the sphenoid bone which forms the anterior boundary of the middle cranial fossa (temporal line). Dodd *et al.*[1] have named these the "triple line shadows" and have emphasized the importance of recognizing these shadows and establishing their integrity roentgenologically.

On occasion, one has difficulty in identifying and distinguishing the "antral line" and the "orbital line" on routine examination. Dodd *et al.*[1] recommend a special angled view of the base of the skull when this difficulty arises.

Believing that a thorough analysis of the anatomy of the orbit and antrum would permit proper roentgenologic identification of the lines, a study, using roentgenograms of a dried skull marked with opaque wire as well as many routine submentovertical skull roentgenograms, was undertaken. An attempt was made to duplicate the varying projections which are employed in making routine base roentgenograms of the skull.

As a result of this work, it is felt that the "antral" and "orbital" lines can be readily distinguished and studied on routine submentovertical roentgenograms in almost all instances.

METHOD

Opaque wires were placed on a dried skull delineating the orbital portion of the great wing of the sphenoid bone which forms the lateral wall of the orbit (the "orbital line") (A). The sphenozygomatic suture (B) which is the anterolateral extremity of the great wing was also marked. The lateral wall of the maxillary antrum (the "antral line") (C and E) and the anterior portion of the great wing of the sphenoid which bounds the middle cranial fossa (the "temporal line") (D) were likewise marked with wires.

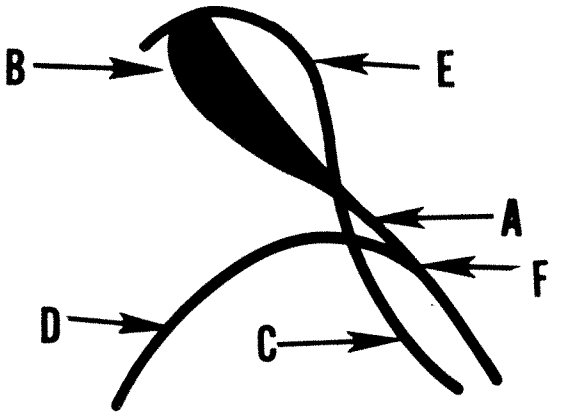


FIG. 1. Drawing of the "triple line shadows" as seen in a submentovertical projection. A. Orbital line; B. sphenozygomatic suture (protuberance); C. posterior portion of antral line (convex laterally); D. temporal line; E. anterior portion of antral line (concave laterally); and F. fusion of orbital and temporal lines. (All figures are marked similarly.)

Submentovertical roentgenograms of the skull were made, varying from the optimum projection in which a true base view is obtained to the less satisfactory projection which approximates an exaggerated Waters' view. These roentgenograms were studied and a description with accompanying illustrations showing the anatomic features which identify the structures is presented.

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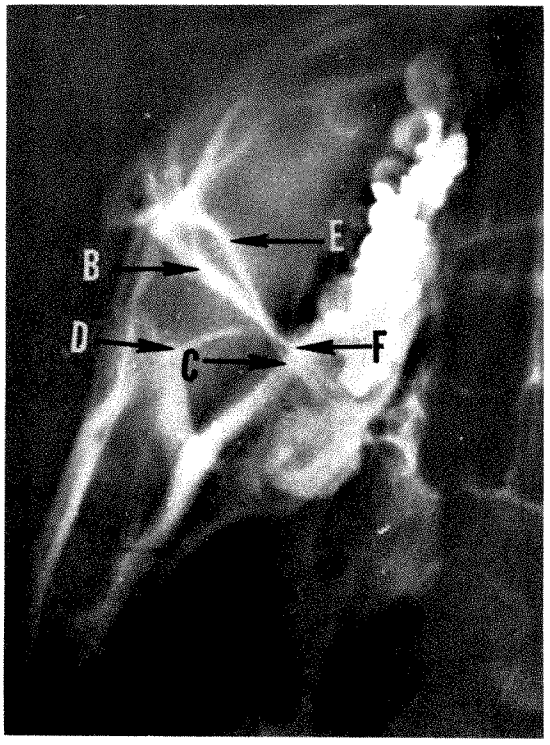


FIG. 2. Submentovertical roentgenogram of the right side in a normal patient.

DISTINGUISHING CHARACTERISTICS OF THE LINES (Fig. 1–5)

THE LATERAL WALL OF THE BONY ORBIT ("ORBITAL LINE")

1. The anterior portion of the "orbital line" (A) is characterized by a bony protuberance which represents the sphenozygomatic suture (*B*). The great wing of the sphenoid is thickened at this site where the anterior portion of the temporalis muscle originates. This prominence is readily identifiable roentgenographically.

2. The posterolateral wall of the orbit and the anterior bone margin of the temporal fossa fuse because they are both portions of the great wing of the sphenoid bone (F). Therefore, the "orbital line" (A) fuses with the curvilinear "temporal line" (D) posteriorly (F).

THE LATERAL WALL OF THE MAXILLARY ANTRUM ("ANTRAL LINE")

1. Anteriorly and laterally the "antral line" curves smoothly to join the zygoma. This curve is concave laterally (E).

2. The "antral line" curves medially at its posterior aspect to join the posterior antral wall. This portion of the curve is convex laterally (C). These two curves give the "antral line" an "S" or reverse "S" shape of varying prominence (C and E).

3. The "antral line" has no bony prominence at its anterior extremity and it does not join with the "temporal line" posteriorly.

DISCUSSION

The relative positions of the "antral line" and "orbital line" are of no practical sig-

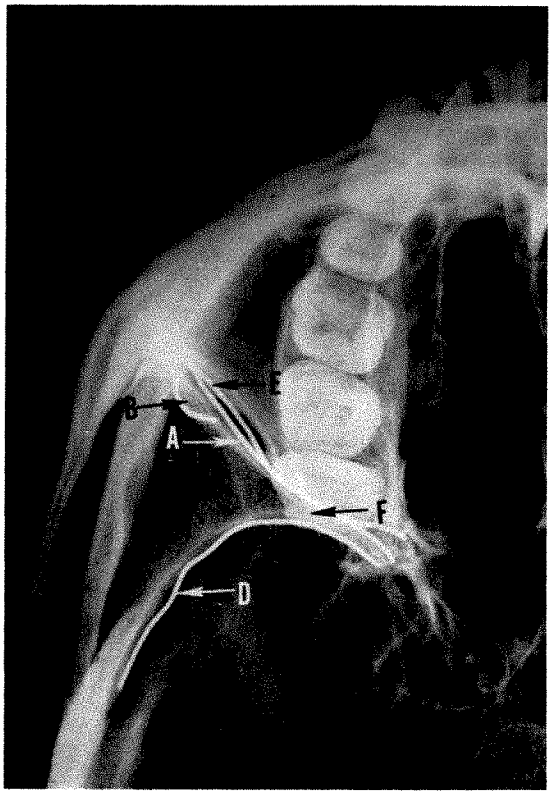


FIG. 3. Submentovertical roentgenogram of the right side of a dried skull. The mandible was removed. Wires have been applied to the bones which cast the triple line shadows. The sphenozygomatic suture was labeled with opaque wire (B) and corresponds to the protuberance described. Note that the antral line (E) lies anterior to the orbital line (A). In this projection the posterior portions of the lines tend to overlap.

nificance as far as identification is concerned (Fig. 3–5). The position of the patient and the angle of the tube when the films are exposed determine the relative positions of the lines. Because the "antral line" is caudad to the "orbital line", submentovertical roentgenograms which approach the Waters' view will show the "orbital line" anterior to the "antral line" throughout their course (Fig. 4 and 5).

In a true supero-inferior projection, the major portion of the "antral line" lies anterior to the "orbital line" and the posterior portions of the lines tend to overlap (Fig. 3). The opposite occurs in the Waters' view, *i.e.*, the anterior segments of the lines tend to overlap (Fig. 4 and 5).

SUMMARY

The roentgenologic and anatomic features of the "orbital" and "antral" lines were determined by study of clinical roentgenograms and roentgenograms of a marked dried skull. These details are described and illustrated.

Knowledge of these facts rather than the relative positions of the lines will permit their proper identification on almost all routine roentgenograms of the skull in the supero-inferior projection.

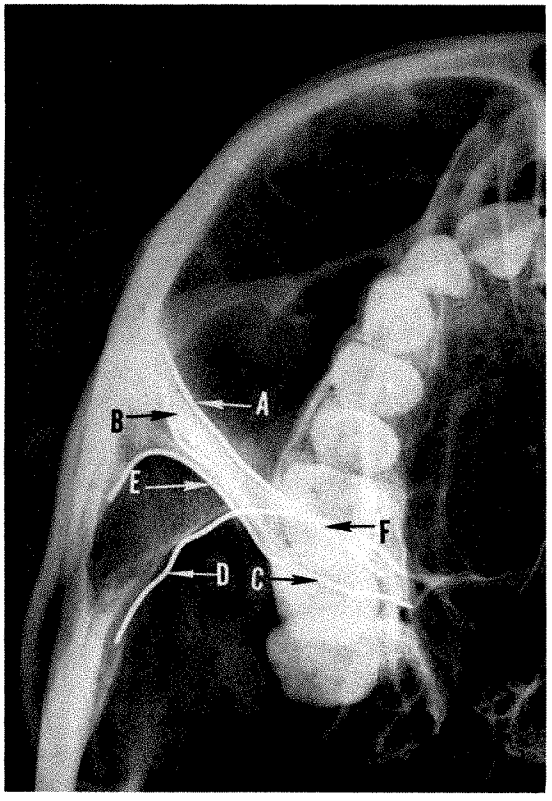


Fig. 4. Suboptimal submentovertical roentgenogram of the right side of a dried skull (mandible removed). Note that antral line lies posteriorly. The anterior ends of the antral and orbital lines tend to overlap. Note that the orbital and temporal lines remain fused (F).

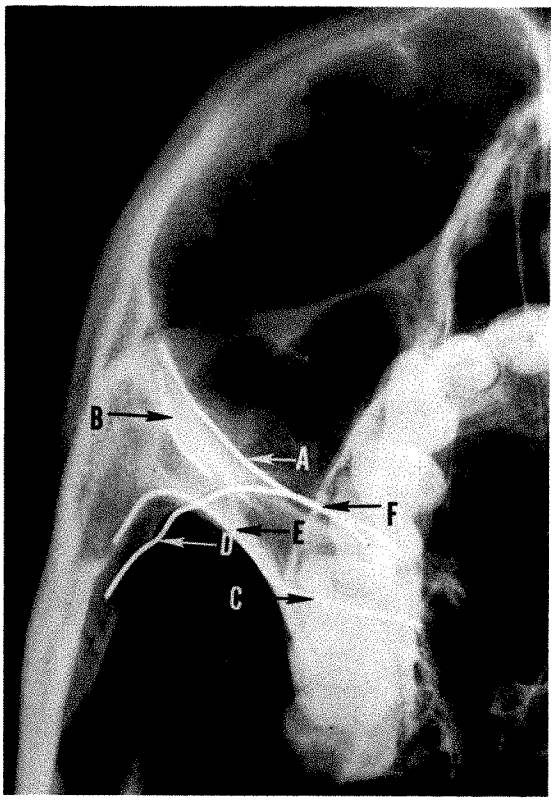


Fig. 5. Base roentgenogram approaching Waters' view of the right side of a dried skull (mandible removed). Note that the antral line lies entirely posterior to the orbital line. The orbital and temporal lines remain fused (F).

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OSSIFYING FIBROMA OF THE FACE AND SKULL*

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OSSIFYING fibroma, an entity of controversial nature, comprises a rather uncommon, localized and slow growing tumor which is found mainly in the maxillae and calvarium of children. After the comprehensive report of Sherman and Sternberg[5] in 1948 dealing with the roentgenologic findings of this tumor, we find several reports of the same entity under other names such as fibro-osteoma[8] and fibrous dysplasia.[4,5,7]

There are some pathologic as well as roentgenologic features of this tumor which make it different from others found in the face and skull. A brief review of such features in this paper may aid in the differential diagnosis of ossifying fibroma from other tumors of fibrous characteristics occurring at the same sites.

CLINICAL AND PATHOLOGIC FEATURES

The ossifying fibroma presents itself as a sharply circumscribed bone tumor which produces a smooth, roundish mass about the orbit, maxillary sinuses or mandible. It arises from the normal bone and becomes a hard tumor firmly attached to the underlying structures. It grows slowly and painlessly, occasionally encroaching upon the nasal airways producing obstructive symptoms. It is usually localized and unilateral but there are cases where the tumor extends itself considerably into the surrounding structures. Rarely, the tumor may undergo sarcomatous degeneration.

When the tumor is studied early, the microscopic examination reveals immature, irregularly calcified and fibrous bone trabeculae. These are interspersed in proliferative connective tissue.[2] Later, a dense fibrous stroma with basophilic bodies appears, forming a framework for bony

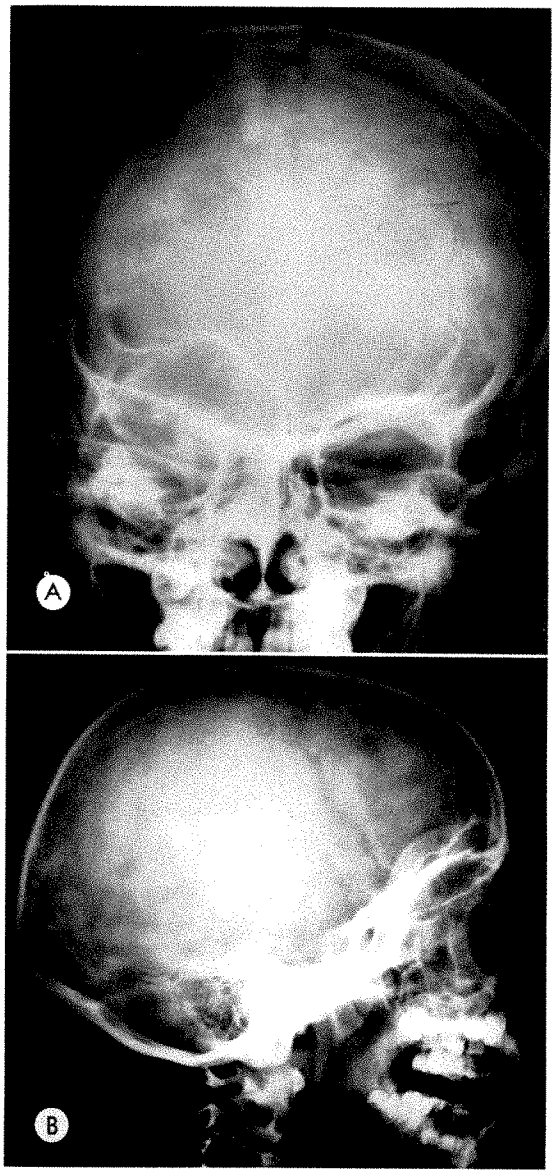


FIG. 1. (*A* and *B*) Nine year old girl with progressive swelling of the right supraorbital area and bulging of the eye of 4 years' duration. The diagnosis of ossifying fibroma was established after surgical removal. Note the smooth, homogeneous bulging of the orbital roof into the anterior cranial fossa.

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islands. These are surrounded by osteoid and the trabeculae which are calcified to various degrees show a few osteocytes. Mitotic figures are absent.[1]

The tumor is surrounded by a thin capsule formed by lamellae of mature bone. The rich vascularity of these tumors has been demonstrated by arteriography. The vessels appear dilated and displaced at the tumor site. There is no formation of arteriovenous fistulae.[8]

ROENTGENOLOGIC MANIFESTATIONS

Ossifying fibroma appears on roentgenograms as a round or oval shaped tumor, clearly circumscribed by smooth margins and producing moderate expansion of the involved portion of bone. It is frequently seen in the frontal bone at the orbital roof. The bone appears spongy at the tumor site with a homogeneous "fibrous" looking pattern and a thin "egg-shell" type of boundary. It extends smoothly into the confines of the cranial fossa forming an even prominence resulting from the expanded and thinned out orbital roof. There is no breakage of the cortical bone as a rule. No bone reaction is found to occur when the tumor is first discovered and the distortion of the surrounding bone is minimal (Fig. 1, *A–D*). In other instances the tumor appears sclerotic, producing thickened, dense bone that surrounds the tumor and extends into the neighboring structures (Fig. 2). This variant is commonly diagnosed as fibrous dysplasia because of its distinctive sclerotic appearance. In adults, this variant could represent the tumor sometimes diagnosed as osteoma (Fig. 6, *A* and *B*).

A combination of these two types is noted when the tumor is located in the mandible. Areas of sclerosis alternate then with smooth and spongy looking bone which expands slowly as the tumor grows, producing deformity and displacement of the teeth (Fig. 3).

In the maxilla, the ossifying fibromas obliterate the antral cavity and disrupt the normal architecture of its boundaries. Expansion of the bone occurs at the expense of

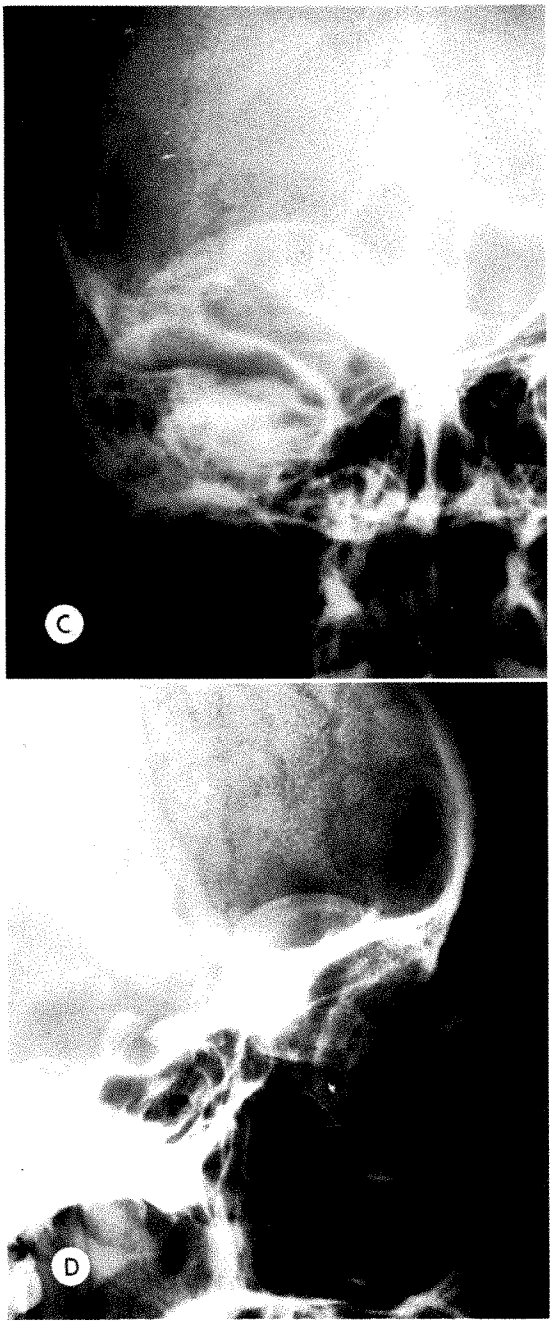


Fig. 1. (*C* and *D*) Very similar findings as in Figure 1, *A* and *B* are noted on these roentgenograms of an 18 year old girl with occasional headaches, swelling and protrusion of the right eye of 6 months' duration (trauma to the area was suffered 6 months prior to the onset of symptoms). A lesion was removed from between the two layers of the orbital roof through a frontal craniotomy. Microscopic study revealed the features of ossifying fibroma.



Fig. 2. Ossifying fibroma was diagnosed after surgery in this young boy with frontal bulging. Note the sclerosing tendency which occasionally occurs in this type of tumor.

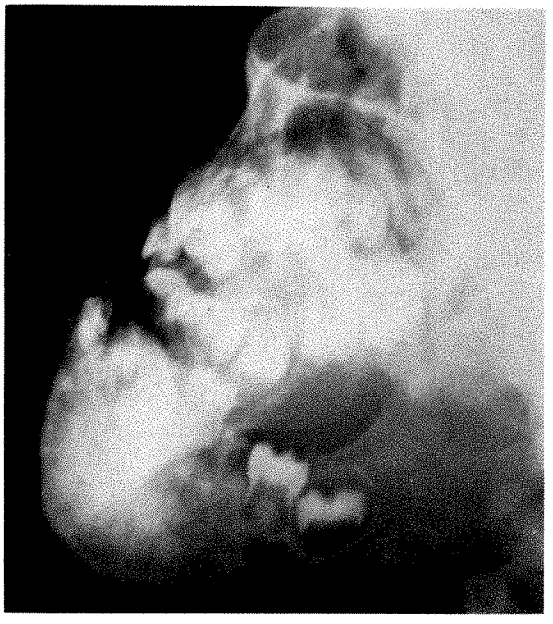


Fig. 3. A combination of the two roentgenologic characteristics of this tumor is shown in the mandible of this child. The pathologic features are those of oss fying fibroma. Spongy and sclerosing bone is seen in the lesion.

the site of least resistance and thus ballooning out through the confines of the outer aspect of the maxilla occurs into the region of the cheek and perhaps extending into the zygoma (Fig. 4). Other times it may be localized to the antral area with a discrete superior extension producing a smooth bulge at the floor of the orbit, with marked thinning but no breakage of the cortical bone of the inferior orbital rim (Fig. 5).

DISCUSSION

The identification of a smooth, clearly circumscribed and homogeneously dense bone tumor, without unusual sclerosis or bone producing characteristics should be suspicious of the presence of an ossifying fibroma, especially if it is found in its preferred locations: frontal bone, maxilla and mandible. This tumor may represent a fibrous type of reaction to certain stimuli as pointed out by Aegerter and Kirkpatrick.[1] This reaction may be set up by external causes such as trauma, local ischemia, hemorrhage or pressure producing hematomas. It is always monostotic and tends to confine itself to a limited area of a membranous bone, arising usually at a center of ossification,[3,6] but showing no cartilage on

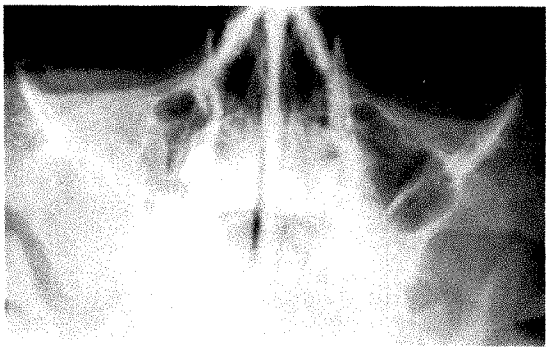


Fig. 4. The expanding characteristic of the tumor is found in this case, an 8 year old boy with a mass in the right maxilla of 3 years' duration. Note the extension of the lesion which partially obliterates the antrum and grows outward, involving the zygoma. The lesion is dense, homogeneous and shows no break in the cortical areas.

microscopic study. Its occasional tendency to sclerose with extension to the surrounding bone makes it impossible to differentiate from monostotic fibrous dysplasia; but its clinical appearance, solitary nature and roentgenologic features make it distinct from other fibrous bone tumors.

CONCLUSIONS

The awareness of the radiologist in diagnosing ossifying fibroma of the skull and face should be dictated by a strict correlation between the clinical findings and the very suggestive roentgenologic features. The final and definite diagnosis should be left to the pathologist.

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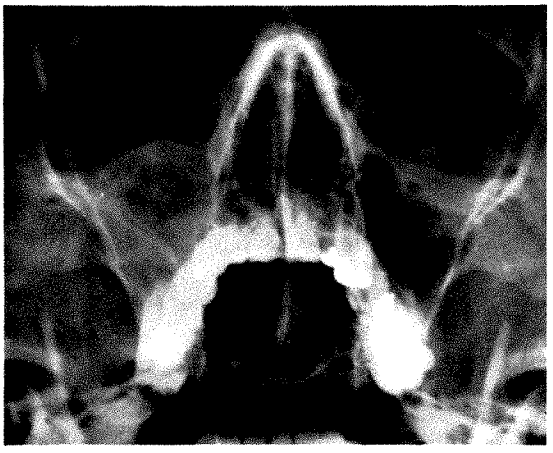


Fig. 5. Ossifying fibroma occurring in a 20 year old patient with pain and swelling below the right eye of 3 years' duration. The diagnosis was made after surgical removal. Note the smooth bulging with encroachment of the inferior orbital area. The bony cortex is intact at the orbital rim.

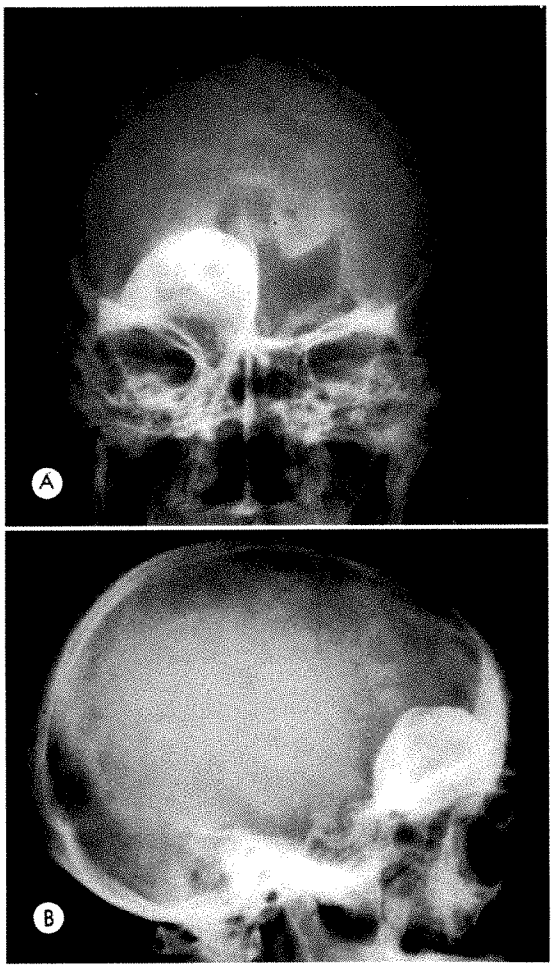


Fig. 6. (*A*) Anteroposterior and (*B*) lateral views of the skull of a 60 year old patient with a diagnosis of osteoma. Note the similarity of the roentgen findings with those in the previous figures. It is possible that an ossifying fibroma may go undetected for many years. A superficial biopsy may show only the osteoma type tissue present in the cortex.

OSTEOCARTILAGINOUS TUMORS OF THE BASE OF THE SKULL*

REPORT OF A UNIQUE CASE AND REVIEW OF THE LITERATURE

By TRYGVE O. GABRIELSEN, M.D.,† *and* LT. COL. ALLEN F. KINGMAN, JR., MC, USA‡
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INTRACRANIAL neoplasms of cartilaginous origin are rare.[10,15,16,21,24] The authors recently encountered a case in which an osteochondroma arose from the posterior surface of the petrous portion of the temporal bone, lateral to the cerebellopontine angle. Because a review of the literature[1–30,32,33] has disclosed no previous report of a similar lesion originating in this location, publication of the case seems warranted. Some pertinent clinical, pathologic, and roentgenographic features of osteochondromas and chondromas of the base of the skull will also be discussed.

REPORT OF A CASE

This 31 year old white female was first admitted to Brooke General Hospital on March 16, 1962 with a 7 year history of visual difficulty characterized by quivering of images, particularly on looking to the right. Two years prior to admission she developed progressive weakness and incoordination of the right extremities. A diagnosis of multiple sclerosis had been made elsewhere at that time. Occasional tingling in the left hand was of 6 months' duration. In the 3 weeks before hospitalization, the patient noted tightness in the posterior cervical musculature but no headache. Because of symptom progression, she again sought medical attention. Roentgenograms of the skull were taken for the first time, with the finding of posterior fossa "calcification," and the patient was referred to Brooke General Hospital.

Physical Examination. The abnormalities were limited to the nervous system. The gait was ataxic, most marked on tandem walking, and there was ataxia of the right extremities, principally in the arm and hand. Rotary nystagmus was present on right lateral gaze, with a fine horizontal nystagmus on all other gazes. The remainder of the examination including audiometric and caloric studies were normal.

Roentgenographic Examination. On the basis of roentgenograms of the skull, a diagnosis of osteochondroma of the base of the skull was made.

Routine posteroanterior studies of the skull showed the lesion largely superimposed over the dense shadows of the petrous ridge and orbital roof, and the tumor was, to a great extent, also hidden from view behind the petrous and mastoid divisions of the temporal bones in lateral projections. The occipital roentgenogram of the skull (Fig. 1) and laminagrams (Fig. 2 and 3) were most helpful.

A large, bony or calcified mass with irregular radiolucent areas within it appeared to arise in a cauliflower fashion from the posterior surface of the petrous portion of the right temporal bone. The intracranial part of the growth was thought to be confined to the posterior cranial fossa. In its attachment to the base of the skull, the medial extent of the tumor seemed to be near the internal auditory meatus. The lesion extended laterally to about one-half inch from the lateral cranial vault, near the sinodural angle.

The left lateral dural sinus appeared to be well developed, whereas there was no definite grooving in the occipital bone from a right lateral sinus. A skeletal survey revealed no additional osteocartilaginous growth.

Interval Course. Because of the large size of the lesion and its benign character together with the patient's rather minimal disability, it was at first decided that surgical removal should not be attempted, and the patient was discharged.

The patient remained unchanged until June

* This material has been reviewed by the Office of The Surgeon General, Department of the Army, and there is no objection to its presentation and/or publication. This review does not imply any endorsement of the opinions advanced or any recommendation of such products as may be named.

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Fig. 1. An occipital roentgenogram of the skull demonstrates a large, osseous or calcified mass arising from the petrous portion of the right temporal bone. The medial border of the lesion is thought to be near the internal auditory meatus.

29, 1962. While coughing, due to an upper respiratory infection, she developed a sudden, severe headache at the vertex followed by a throbbing ache behind the orbits and in the face. She was re-admitted to Brooke General Hospital on June 30, 1962 in an acutely uncomfortable state. In addition to the previous neurologic abnormalities, funduscopic examination showed venous engorgement and 1 diopter papilledema on the right. Mild right facial weakness was present.

Operative Findings. The lateral ventricles were found to be markedly enlarged on introducing a catheter for ventricular drainage. Suboccipital craniectomy revealed a completely extradural, bony mass of varying density arising laterally just deep to the mastoid process, with the base extending medially along the posterior aspect of the petrous pyramid. The mass filled one-fourth of the posterior cranial fossa. Piecemeal removal was carried out by both intradural and extradural approach. The tumor contained regions of yellowish, soft, myxomatous appearing material. It was possible to remove all of the intracranial portion of the mass, leaving a base measuring approximately 2.5 cm. in diameter on the posterior aspect of the petrous and mastoid portions of the temporal bone (Fig. 4). The sigmoid sinus was

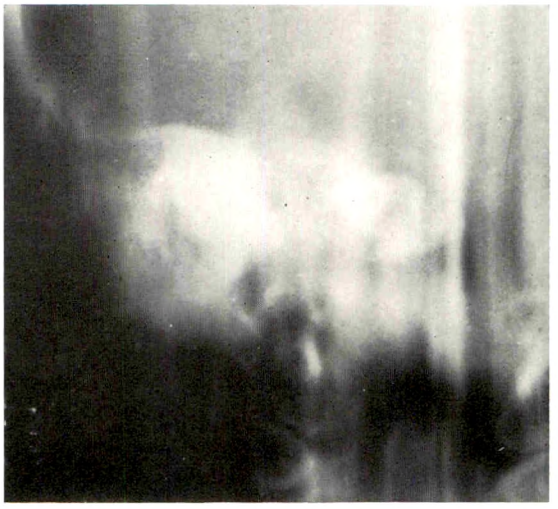


Fig. 2. An anteroposterior laminagram through the base of the mass shows a bony defect where the lesion is attached to the underlying normal bone. This defect is most likely due to noncalcified cartilage.

not identified and was apparently obliterated from long standing extradural pressure. An anatomic variant could perhaps also be responsible. The cerebellar tonsils were herniated through the foramen magnum to the level of C2.

Pathologic Findings. (Examination by Capt.

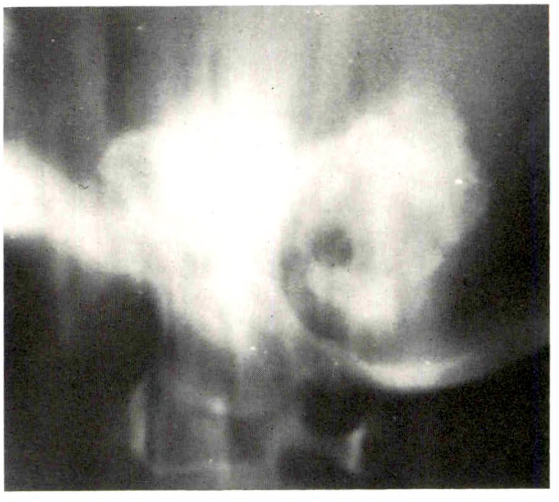


Fig. 3. A lateral laminagram demonstrates to good advantage the cauliflower appearance of the mass, which arises from the posterior aspect of the petrous portion of the temporal bone. There are areas of relative radiolucency within the predominantly dense shadows from bone or calcified cartilage.

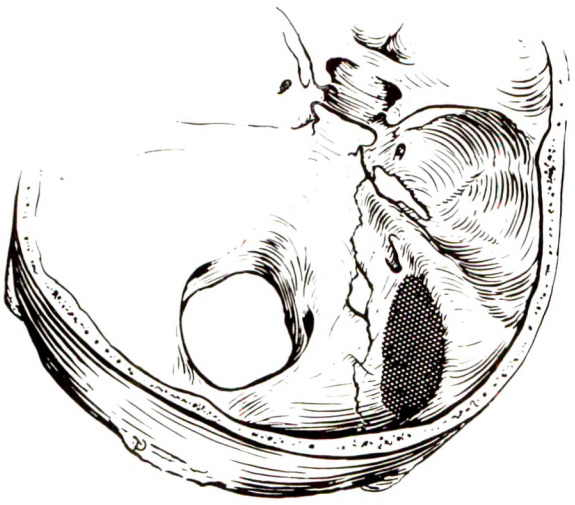


Fig. 4. Artist's drawing showing the estimated base of the lesion at the time of surgery.

J. M. Hardman, MC, USA.) After 10 per cent buffered formalin fixation, the tissue was decalcified and stained with hematoxylin and eosin. Microscopically, the lesion removed from the right petrous bone had a cap of mature bone and a core of mature cartilage. The bone was randomly arranged with thick trabeculae surrounding fatty spaces devoid of hematopoietic cells. The trabeculae were more closely approximated near the surface and were universally traversed by mosaically arranged cement lines. The eosinophilic osteoid surrounded lacunae filled with single, uniform unremarkable osteocytes. Near the core, cartilage was intermixed with bone (Fig. 5). The cartilaginous areas had an eosinophilic hyaline matrix surrounding lacunae filled with single, uniform unremarkable chondrocytes. Focally, the cartilaginous matrix resembled loose fibrous tissue with or without slight myxomatous degeneration. Centrally, the lesion contained similar, mature, slightly vacuolated, unremarkable cartilage.

Follow-Up. Postoperatively, the patient did well but initially showed increased right facial weakness and hypalgesia in all divisions of the V cranial nerve. There was disorganization of conjugate eye movements in all directions and increased nystagmus. Ataxia was likewise increased. These findings all gradually improved, and she was discharged ambulatory 18 days following surgery. At that time extraocular movements were normal except for rotary nystagmus on right lateral gaze, of the same degree noted preoperatively. The ataxia was also essentially the same as prior to the operation.

Following discharge from the hospital, the patient showed further improvement in the extremity and truncal ataxia to a state better than existed immediately preoperatively. Definite disturbance remained 5 months later and did not change during the succeeding 3 months. Rotary nystagmus on right lateral gaze remained unchanged, together with decreased pain perception in all divisions of V cranial nerve on the right, but there was no disturbance of touch. The right facial weakness cleared completely within 2 months following surgery. At last follow-up, the patient was pregnant.

DISCUSSION

CLASSIFICATION

Intracranial neoplasms containing cartilage may occasionally be found in the presence of similar lesions elsewhere in the skeleton or even associated with soft tissue hemangiomas.[10,16,21,30] Whereas the former have a distinct familial tendency, there is no convincing evidence that solitary osteochondromas and chondromas are hereditary.[5,14,16,17]

Osteocartilaginous growths apparently originating from choroid plexus or meninges of the brain adjacent to the cranial vault have been attributed to metaplasia.[8] There is no demonstrable connection with the cranium in these lesions.[16,17] The theory has also been advanced that some of the reported cases may represent unusual ossifying meningiomas.[16]

Cartilage-containing tumors of the paranasal sinuses on rare occasions become manifest secondarily as an intracranial mass, especially at the floor of the anterior cranial fossa. They probably have a similar origin as chondromas and osteochondromas arising in a primary site at the base of the skull. Development from cartilaginous rests retained along the basilar synchondroses appears likely,[16,21] since the bones at the base of the skull are derived from the chondrocranium.[24,31]

We have gained the impression that authors writing in English have variously

called cartilage-containing tumors at the base of the skull either chondromas or osteochondromas, depending on the histopathologic appearance. These growths all seem to be regarded as chondromas in the German literature, undoubtedly due to their generally accepted common origin from embryonic cartilaginous rests. Our case of osteochondroma is thought to belong in this group of neoplasms.

All of the preceding lesions should probably be considered separate from the osteomas, which generally present in the frontal sinuses or on the external aspect of the skull. These include ivory exostoses and spongy osteomas. Jaffe[14] states that an ivory exostosis ordinarily develops in relation to a bone pre-formed in membrane rather than cartilage. The spongy type osteoma appears to favor a similar location but tends to be less uniformly dense and larger than the ivory exostosis.[24]

INCIDENCE

The rarity of intracranial, cartilaginous neoplasms is demonstrated by the fact that Leitholf[20] found only 4 chondromas among 4,135 brain tumors seen at the Serafimerlasarettet in Stockholm. Of more than 6,000 brain tumors at the Tönnis Clinic in Cologne, there were evidently 9 classified as chondromas of the basilar synchondroses.[3,16,17,22] Kleinsasser[16] maintains that these tumors are encountered even less frequently in this location than in paranasal sinuses. In 1960 he estimated that only 30 to 35 such cases had been described in the world literature. We believe that several lesions reported by the original authors[15,18,21] as osteochondromas of the base of the skull are included in this estimate.

LOCALIZATION

Osteochondromas and chondromas of the base of the skull seem to be extradural in origin but may sometimes erode through the dura mater. They most frequently present as a middle cranial fossa mass, particularly in the parasellar region, where so many of the cranial sutures converge.[16,17] Some of the published cases have been extremely large neoplasms, and the exact site of origin has been difficult to establish. In some such instances, the mass involved both middle and posterior cranial fossae.* It appears that the very few basilar chondromas and osteochondromas reported to originate in the posterior cranial fossa have been located in the cerebellopontine angle or region of the petrous apex.[1,3,6,10,17,18,29]

The current case report has been submitted because the osteochondroma in our patient definitely arose in a more lateral location. In its attachment to the cranium only the most medial extent of the lesion



Fig. 5. Microscopic appearance of the excised specimen, which reveals the presence of both bone and cartilage. This section was taken from a considerable depth within the tumor.

* A summary of cases published prior to 1951 can be found in Klingler's[15] extensive review of intracranial, cartilaginous tumors at the base of the skull.

reached the posterior border of the internal auditory meatus.

The petromastoid portion of the temporal bone develops from the chondrocranium as four separate ossification centers, which subsequently fuse.[31] The authors cannot conclusively correlate one of these sutures with the site of origin of the osteochondroma described in the present report. The tumor arose from the temporal bone above the petro-occipital suture (Fig. 4).

CLINICAL FEATURES

Chondromas and osteochondromas of the base of the skull show some predilection for females and occur mostly from the second through the fifth decade of life.[16] The clinical symptoms may be surprisingly scanty even when the lesion is quite large.[4,15] Our patient had symptoms referable to the tumor for at least 7 years. The relative lack of neurologic abnormality undoubtedly can be explained on the basis of very slow growth of the neoplasm. Such a protracted clinical course was also noted by List.[21]

Although these lesions are benign histologically, they usually have a serious implication because of their critical location.[16] The ultimate prognosis is poor.[18]

The operative removal of the mass is often extremely difficult, due to the intimate relation of the tumor to cranial nerves, large intracranial arteries, or vital centers in the brain stem.[22] Total extirpation of the lesion is certainly not the rule.[16,21] However, even a subtotal excision will often give the patient many additional years of useful life.[17,21]

A relatively rapid growth or early postoperative recurrence should raise the suspicion of chondrosarcoma.[14,16,17,24] Although a histologic diagnosis of chondrosarcoma would indicate an ominous prognosis for the patient, one should not necessarily anticipate a short survival time.[18]

PATHOLOGIC FINDINGS

An osteocartilaginous exostosis should preferably have at least a thin surface cap of cartilage, which may be difficult to identify and may virtually disappear after growth of an osteochondroma has ceased.[5,14] Such a cartilaginous rim could conceivably have been missed in our case, since it was necessary to remove some of the tumor piecemeal by rongeur.

On the other hand, it should be noted that significant amounts of cartilage were identified deep in the main specimen submitted for microscopic examination (Fig. 5). The case described by King and Butcher[15] apparently showed similar findings. They commented that the cartilaginous portions in osteochondromas as a rule lie superficial to the bone elements and were struck by the reversal of the usual relationship in parts of the tumor.

These observations might lend further support to Kleinsasser's[16] statement that most of the so-called osteochondromas at the base of the skull may be chondromas with bone formation. Chondromas are certainly known to show ossification.[5,14,33] The published cases of intracranial, cartilaginous growths have contained widely different proportions of bone and cartilage. Some have apparently revealed no osseous elements and must clearly be chondromas.[1,18]

However, both Jaffe[14] and Dahlin[5] say that islands of cartilage are at times embedded in the underlying cancellous bone of an osteochondroma. Consequently, the mere presence of cartilage deep in the spongy section of a bone tumor should not in itself exclude the diagnosis of osteochondroma.

Extracranial, osteocartilaginous exostoses grow in a fairly organized fashion by endochondral ossification on the inner surface of the cartilage cap.[14] This process admittedly was not demonstrated in the neoplasm studied by the authors and has probably not been adequately documented in pertinent previous case reports either. Kleinsasser's[16] observation that extracranial osteochondromas tend to have a more characteristic, organized, architectural pattern than the intracranial ones may be significant. Nevertheless, the authors have found no definite objection to

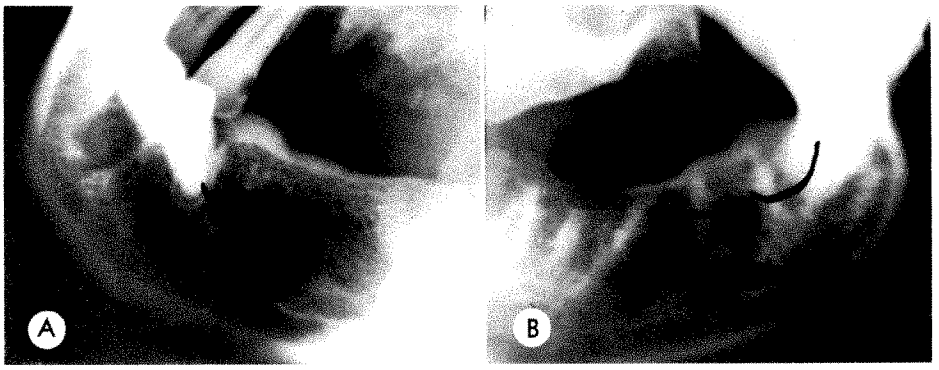


Fig. 3. (*A*) Right and (*B*) left torus, demonstrated by lateral oblique exposure only because of edentia.

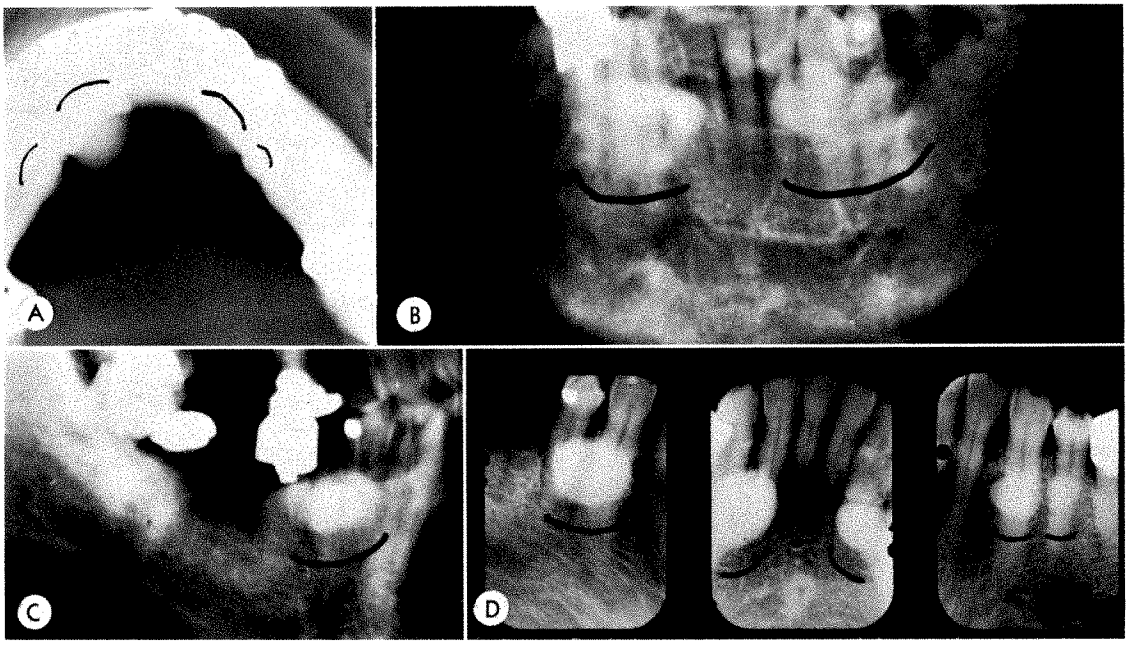


Fig. 4. (*A*, *B*, *C* and *D*) Bilateral tori demonstrated by 4 exposure-postures. The small dental-root roentgenograms are characteristic and should be recognized.

either by excision (reportedly a very easy procedure) or adaptation of the denture design to fit about them. Most authors believe they do not recur after excision.

SUMMARY

Torus mandibularis is a benign overgrowth of cortical bone, creating a clinical problem when its size and position interfere with dentures of the mandible, or when it causes cancerophobia. The best roentgenographic technique for its demonstration is considered.

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EXPERIMENTAL HEAD TRAUMA*

CEREBRAL ANGIOGRAPHIC OBSERVATIONS IN THE EARLY POST-TRAUMATIC PERIOD

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THE cerebral vascular alterations which take place as the result of head trauma are of interest for many reasons. Paramount among them is the role such alterations may play in the development and course of concussion after head trauma. Also, knowledge of the sequential cerebral vascular alterations following trauma, especially as determined neuroroentgenologically, might afford a method for clinically predicting the severity of the cerebral damage and for following its subsequent course.

This report concerns further details of observations made as part of a continuing study of experimental head trauma, a preliminary report of which has already been made.[8] In this communication, special emphasis is placed upon the results of cerebral angiographic studies performed in the interval 15 to 30 minutes after trauma. Additional experiments designed to elucidate observations made on the angiographic study are also reported.

METHOD

Twenty-four monkeys (M. mullata) of approximately 3 kg. weight and one chimpanzee were used. The animals were divided into two major groups: those to be used for control experiments, and those to which head trauma was to be administered. Head trauma was produced by a compressed air gun, using firing pressures ranging from 10 to 50 p.s.i. A cervical collar was fashioned so that the animal could be struck on the head with or without immobilization of the cervical spine. The details of the preparation of the animals for the studies were given in the earlier paper.[8] Using accelerometers connected to the skull, photographic recordings were made from an oscilloscope of the deflection of the head at the moment of impact, from which the actual peak acceleration was calculated. The apparatus for administering the trauma is shown in Figure 1. In monkeys prepared for carotid angiography, the right common carotid artery was catheterized with a No. 16 Intracath. Those animals in which the blood-brain barrier was to be studied received 5 cc. of sodium fluorescein intravenously at least 1 hour before head trauma or angiography.

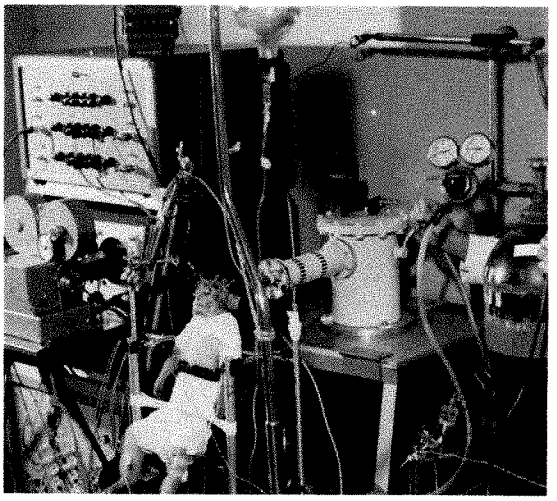


FIG. 1. Apparatus for administering head trauma and recording accelerometric data.

The physiologic data which were recorded on a Grass 8-channel electroencephalographic machine consisted of four channels of electroencephalograms, one of an electrocardiogram (standard limb leads) and one of respiration (with a chest pneumotachometer). In 3 animals, intra-

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arterial blood pressure was recorded on a D.C. strip chart recorder. Of the traumatized animals, 7 were allowed to regain full consciousness after preparation. Trauma was then administered while they were immobilized in a neoprene suit after local analgesia of the region to be traumatized.

Of those animals in which carotid angiography was performed, serialographic roentgenograms were made with the heads in the lateral projection. A Sanchez-Perez film changer, taking roentgenograms at 1 second intervals for 12 seconds, was used. The contrast material (50 per cent hypaque) was injected as rapidly as possible by hand through the carotid catheter. Aside from the control animals, 1.5 cc. of contrast material was generally used per injection, although larger volumes were used in 2 traumatized animals. To allow ease of study of the roentgenograms, the image was magnified by using a 0.3 mm. focal spot in the roentgen ray tube and displacing the animal from the film. All films were processed in a Kodak X-omat Automatic Processing Unit. Plain roentgenograms of the cervical spine and skull were made after trauma in order to determine the presence of skull or cervical spine fracture or dislocation.

All animals were sacrificed within 4 hours after the trauma and the brain and upper spinal cord were removed *en bloc* and note was made of hematomas, fractures, hemorrhages and contusions. Examination under an ultraviolet lamp was performed and areas of fluorescence were noted and photographed. After surface study, cut sections of the brain and cord also were studied.

Of the animals to be used in the control experiments, 1 was prepared in the same fashion as the animals to be traumatized and to be studied angiographically, except that no contrast material was injected and no trauma was applied. This animal was used to determine the effect of carotid ligation and general anesthesia on the blood-brain barrier. The animals to be used for the study of the effect of the contrast material on the cerebral angiograms and the blood-brain barrier were prepared in similar fashion, except that no trauma was administered and multiple injections of contrast material (50 per cent hypaque) were given (Table 1).

RESULTS

From the control experiments performed (Table 1), it was established that the method of carotid ligation and general anesthesia used in this series did not cause any change in the physiologic parameters measured nor did it cause a breakdown of the blood-brain barrier, other than at the expected sites. The results of the control experiments further showed that hypaque 50 per cent, in volumes ranging from 1.5 to 3.0 cc. injected as many as 4 times, caused no alteration of the cerebral circulation time nor in the roentgenographic appearance of the vessels (Fig. 2, *A–D*). The injections of contrast material did cause, however, a breakdown of the blood-brain barrier, but this was primarily in the gray matter of the brain. When repeated doses of 1.5 cc. were used, the breakdown observed was minimal. It is interesting that in 2 animals (M-12, M-16), the breakdown was seen in both hemispheres, even though the injections were unilateral.

In Table II are summarized the data obtained from the traumatized animals. Of the 21 animals in this group, 19 were given occipital trauma and 2 were given trauma to the temporal region. Both animals receiving temporal trauma were given 2 blows each to the same region. Also, two blows were given to one of the animals receiving occipital trauma. The head and cervical spine were free to move during 15 of the blows and immobilized during 9 blows. The accelerations achieved by the blows were found to be dependent upon: the site of the blow (occipital blows created more acceleration than temporal), the force of the blow, and the mobility of the head and cervical spine.

The criteria used for determining the presence of concussion due to the blow were: loss of the responses to external

stimuli including pain; respiratory arrest of over 3 seconds followed by slow irregular respirations and, thirdly, a true bradycardia. In those animals in which concussion was produced under pentothal anesthesia, only the latter 2 criteria were used. Cessation of respiration occurred consistently with concussion, lasted for periods up to 5 seconds, and was then replaced by irregular, often gasping, slow respiration. This abnormal respiration invariably persisted for the duration of unconsciousness. Simultaneously, a marked slowing of the heart rate was consistently noted on the electrocardiogram, often attended by irregularities of rhythm. Concussion invariably resulted in an increased amplitude and slowing of the rhythmic activity on the electroencephalogram, changes whose duration also paralleled the duration of unconsciousness. When the blow did not produce concussion, no significant effects were seen in the electroencephalogram, electrocardiogram or respiration. Utilizing the criteria described, 14 of the traumatized animals were considered to have been concussed while in 7 animals, concussion was not produced, even though 3 of these animals received 2 blows each.

The mechanical factors related to concussion were discussed in detail in our earlier paper.[8] Briefly, these data showed that it is more difficult to produce concussion with the neck fixed than with the head free to move on the neck. This finding, which suggests a protective effect against

TABLE I

RESULTS FROM CONTROL EXPERIMENTS

A. Carotid Ligation and General Anesthesia Control

Monkey No.	EEG	ECG	Respiration	Pathologic Examination
M-14	No change	No change	No change	No B.B.B.B.*

B. Contrast Material Control†

Monkey No.	Injection	Hypaque 50 per cent (cc.)	Cerebral Angiographic Findings	Pathologic Examination
M-8	1	3	Normal angiogram	Not done
	2	3	——	
	3	3	Normal angiogram No spasm, no slowing	
M-9	1	3	Normal angiogram	B.B.B.B. on side of injection in distribution of the middle cerebral artery, gray matter predominantly
	2	3	——	
	3	3	——	
	4	3	Normal angiogram No spasm, no slowing	
M-12	1	1.5	No angiography performed	B.B.B.B. predominantly in gray matter bilaterally
	2	1.5		
M-16	1	1.5	Normal angiogram	B.B.B.B. predominantly in gray matter bilaterally
	2	1.5	Normal angiogram No spasm, no slowing	
M-17	1	1.5	Normal angiogram	Not done
	2	1.5	Normal angiogram No spasm, no slowing	
M-20	1	1.5	Normal angiogram	Not done
	2	1.5	Normal angiogram No spasm, no slowing.	

* B.B.B.B.—blood-brain barrier breakdown.

† M-8, M-17, and M-20 were used for trauma experiments after the control studies.

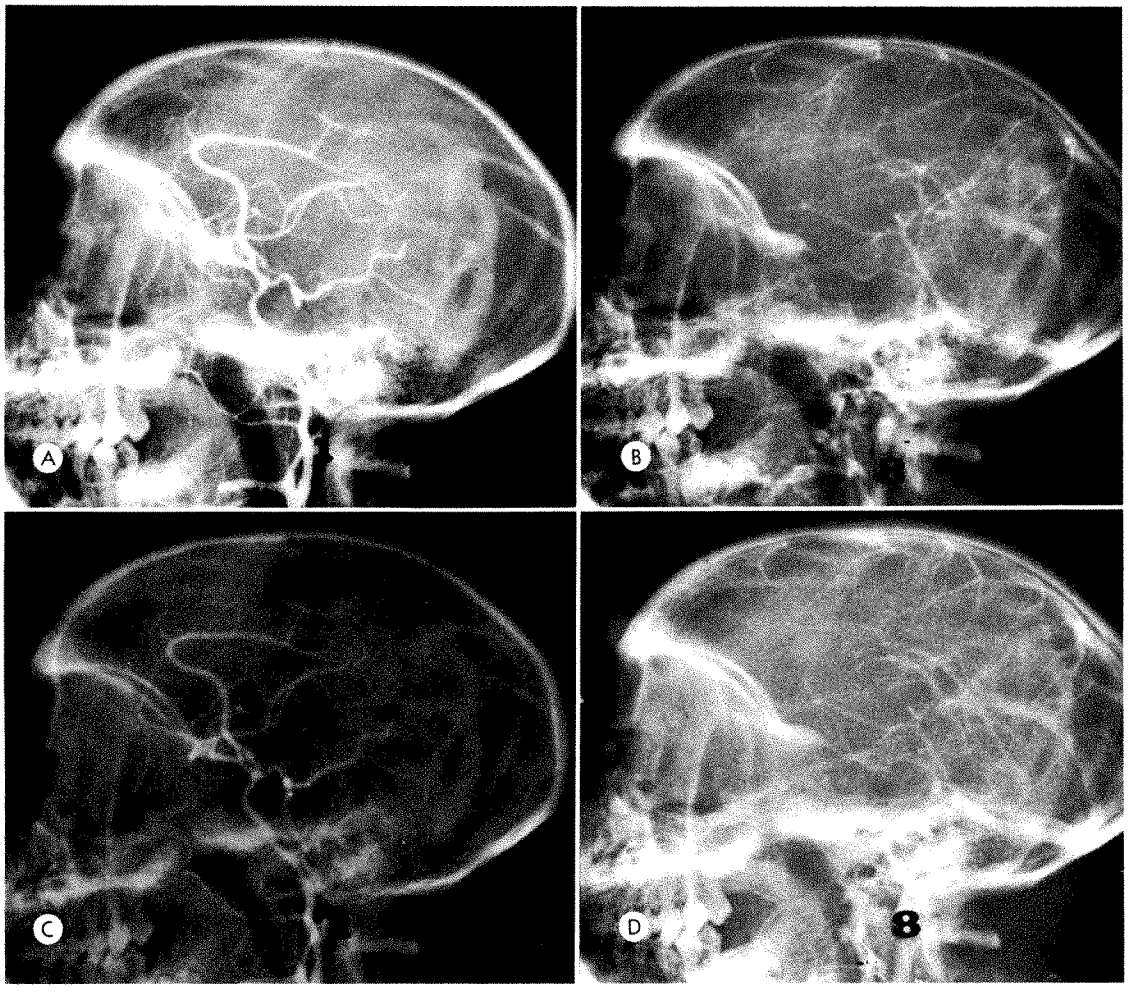


FIG. 2. Control angiograms (M-16, Table 1). *A* and *B* are arterial and venous phase roentgenograms from first injection and *C* and *D* are from the second injection. Numerals refer to time in seconds after injection. Note that injections of contrast material without trauma cause no change in circulation time or appearance of vessels.

concussion afforded by cervical spine immobilization, is strengthened by analysis of the blood-brain barrier breakdown studies (Table II), which showed that occipital trauma with the neck fixed (M-11, M-17, M-24) produced no breakdown in the cervical cord or brain stem while occipital trauma with the head free to move (M-6, 7, 10, 13, 15, 18, 19) uniformly caused breakdown in the brain stem and occasionally in the cervical expansion.

Comparison of the cerebral angiograms made before and after occipital trauma (M-1, 6, 8, 11, 17, 20, 22) in the interval 15 to 30 minutes following the trauma revealed the following findings (Table II):

(a) In all animals in which the circulation time was measured by serialographic roentgenograms (M-8, 11, 17, 20, 22), there was slowing of the cerebral circulation following trauma (Fig. 3, *A–D*). The slowing was observed whether or not skull fracture or sutural diastasis occurred due to the trauma. In one animal in which the calculated acceleration was 3,400 g. (M-22), it is interesting to note that the most profound degree of slowing was observed, the arterial phase lasting 10 seconds (Fig. 4, *A–D*). Although circulatory slowing occurred in this group of animals whether or not concussion

TABLE II

RESULTS FROM TRAUMA EXPERIMENTS

Primate No.	Site of Blow	Air Pressure	Neck	Calculated Acceleration	Concussion Produced	Pathologic and Fluorescein Blood-Brain Barrier Studies	Angiographic Findings	Fractures or Sutural Diastasis
M-1	occipital	20 p.s.i.	Free	500 g.	yes	B.B.B. study not done. Brain normal microscopically one week later	Arteries and veins smaller after trauma. Circulation time not measured	No
M-2	occipital	10 p.s.i.	Free	90 g.	no	B.B.B. study not done. Brain normal macroscopically	Not done	No
M-3 a)	occipital	15 p.s.i.	Free	not recorded	no	Not examined	Not done	No
b)	occipital	15 p.s.i.	Free	not recorded	no			No
M-4 a)	rt. temporal	35 p.s.i.	Fixed	not recorded	no	B.B.B.B.* right temporal and minimum contrecoup	Not done	No
b)	rt. temporal	50 p.s.i.	Fixed	not recorded	no	++ edema on right. Brain stem normal		No
M-5 a)	rt. temporal	50 p.s.i.	Fixed	120 g.	no	Same findings as in M-4	Not done	No
b)	rt. temporal	50 p.s.i.	Fixed	129 g.	no			Right temporal fracture
M-6	occipital	50 p.s.i.	Fixed	930 g.	no	Occipital epidural clot and occipital edema and B.B.B.B.	Post-trauma displacement of sagittal sinus. Circulation time not measured. Probable posterior parietal stain	Sutural separation
M-7	occipital	50 p.s.i.	Fixed	not recorded	yes	B.B.B.B. occipital region, but maximal in brain stem and cervical cord	Not done	Not examined
M-8	occipital	25 p.s.i.	Free	780 g.	yes	B.B.B.B. occipital. Pons contused	Definite prolongation of circulation time post-trauma	No
M-10	occipital	25 p.s.i.	Free	700 g.	yes	B.B.B.B. occipital and brain stem and white matter of fornix	Not done	Sutural separation
C-1†	occipital	20 p.s.i.	Free	300 g.	yes	Not done	Not done	No
M-11	occipital	40 p.s.i.	Fixed	not recorded	no	B.B.B.B. occipital. Normal brain stem	Very slight prolongation of circulation time post-trauma	No
M-13	occipital	50 p.s.i.	Free	1,700 g.	yes	B.B.B.B. occipital and brain stem	Not done	Sutural separation
M-15	occipital	50 p.s.i.	Free	1,850 g.	yes	B.B.B.B. occipital and brain stem	Not done	Sutural separation
M-17	occipital	50 p.s.i.	Fixed	1,100 g.	yes	B.B.B.B. occipital and rt. hemisphere. Normal brain stem	Definite prolongation of circulation time post-trauma	Depressed occipital fracture
M-18	occipital	50 p.s.i.	Free	1,900 g.	yes	B.B.B.B. occipital and brain stem and cervical cord	Not done	Occipital fracture
M-19	occipital	50 p.s.i.	Free	1,950 g.	yes	B.B.B.B. occipital and brain stem and cervical cord	Not done	Occipital fracture
M-20	occipital	50 p.s.i.	Fixed	1,010 g.	yes	Not done	Definite prolongation of circulation time post-trauma	Occipital fracture
M-21‡	occipital	50 p.s.i.	Free	not recorded	yes	Not done	Not done	Not examined
M-22	occipital	50 p.s.i.	Free	3,400 g.	yes	Not done	Marked prolongation of circulation time post-trauma	Occipital fracture
M-23	occipital	50 p.s.i.	Free	technically unsatisfactory	yes	Not done	Technically unsatisfactory	Not examined
M-24	occipital	25 p.s.i.	Fixed	technically unsatisfactory	no	B.B.B.B. occipital and parasagittal. Normal brain stem	Not done	Not examined

* B.B.B.B.—blood-brain barrier breakdown.

† Chimpanzee.

‡ M-21 was used for biochemical studies, to be reported later.

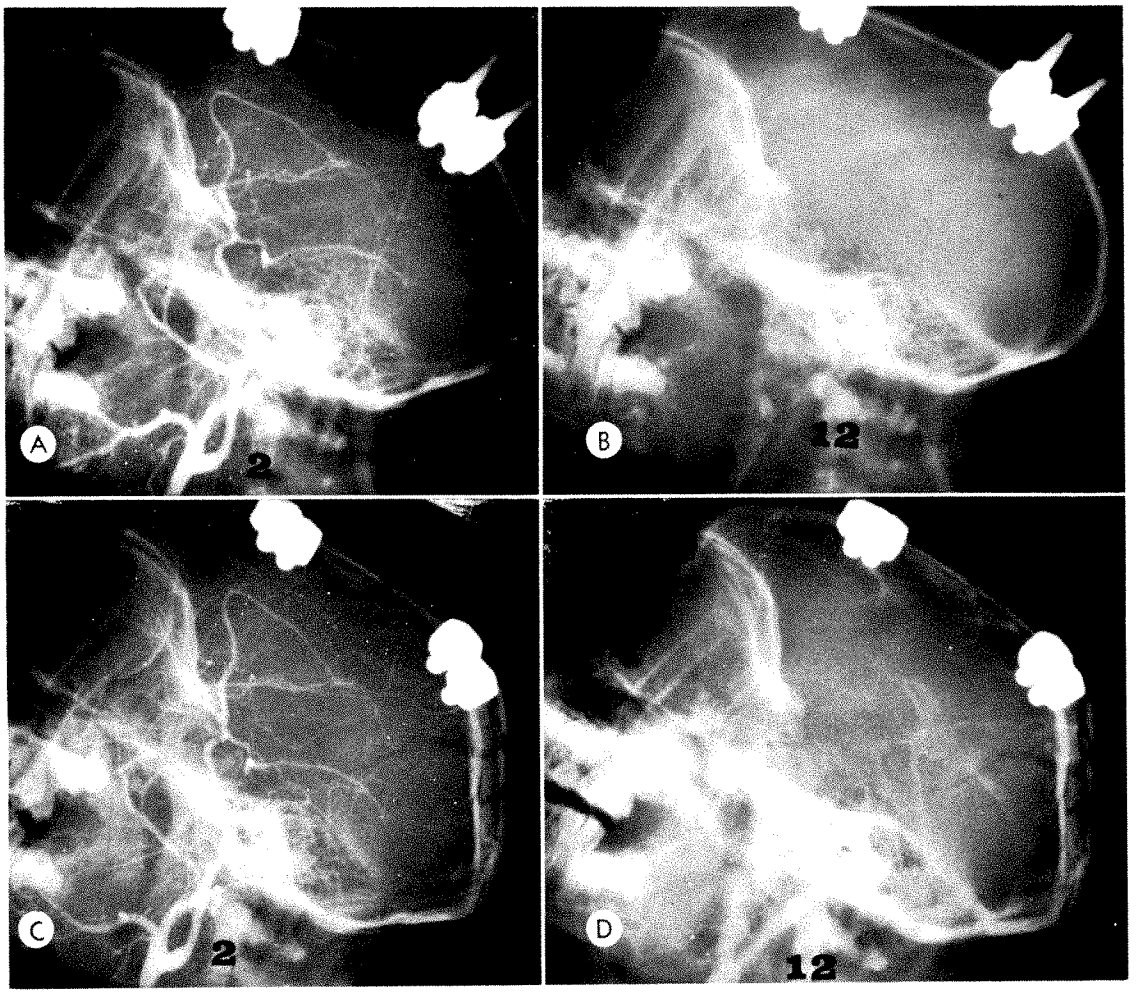


Fig. 3. Slowing of cerebral circulation after trauma (M-1[7], Table II). *A* and *B* are pre-trauma and *C* and *D* post-trauma angiograms. Numerals refer to time in seconds after injection. Note similar appearance of the arteries before and after trauma. Note also the depressed, comminuted parieto-occipital fracture after trauma.

had been produced, in the one animal without concussion in which this was measured, the slowing was very slight.

(b) In one animal (M-6), post-traumatic displacement of the sagittal sinus was seen, in which location at the time of autopsy an epidural hematoma was found (Fig. 5, *A–F*). Also, an apparent posterior parietal stain was noted in the post-trauma roentgenograms. The location of this stain coincided with an area of breakdown of the blood-brain barrier. No stains were seen in any of the other animals, even though blood-brain barrier breakdowns occurred in all of them to some degree. Perhaps the volume of contrast material used to study this animal (4 cc.) was necessary to visualize the breakdown angiographically. The only other traumatized animal receiving more than 1.5 cc. of hypaque for an injection (M-1) did not have a study of the blood-brain barrier performed and no stain was seen angiographically.

(c) In the first animal studied (M-1), definite post-traumatic narrowing of the arteries and veins was observed. This was not observed in any of the other animals. This animal received a larger dose of contrast material than the subsequent animals (5 cc. per injection as opposed to 4 cc. in

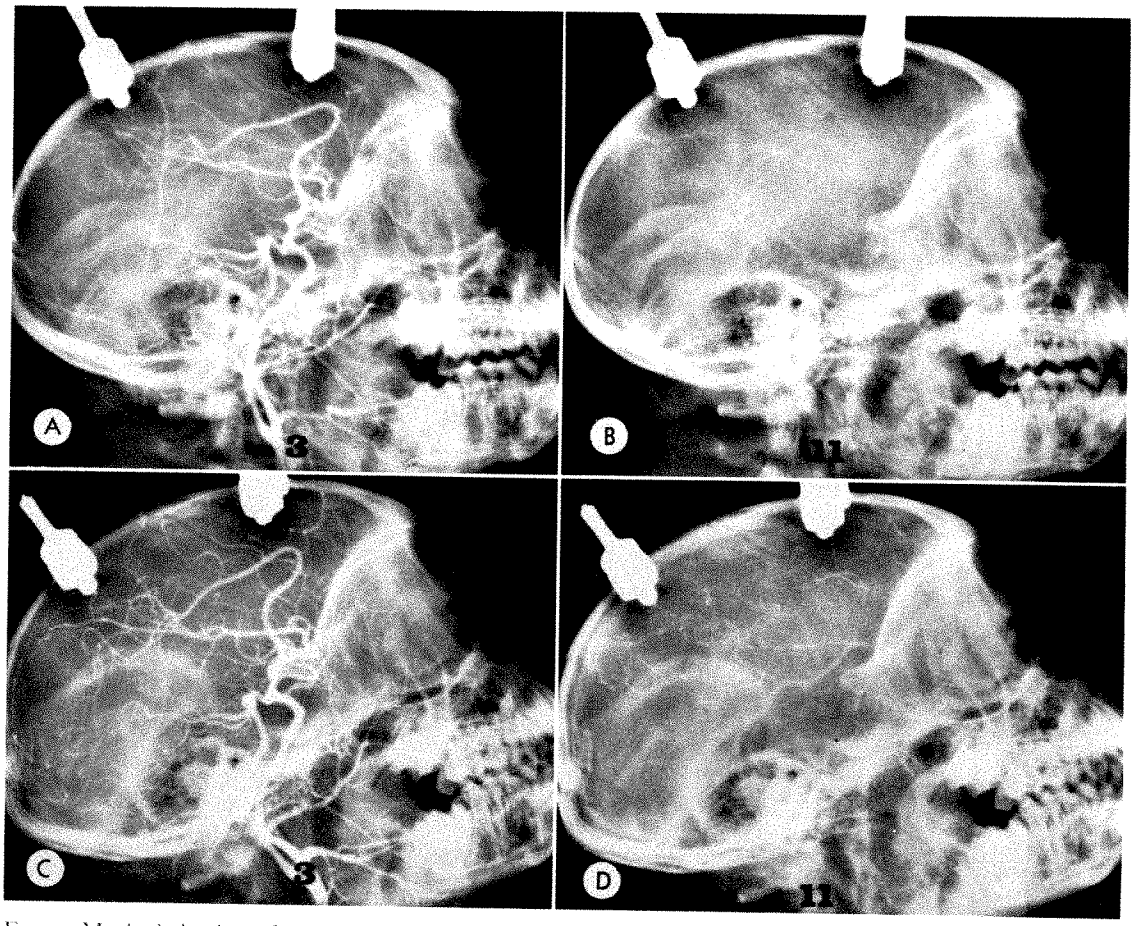


FIG. 4. Marked slowing of cerebral circulation after trauma (M-22, Table II). *A* and *B* are pre-trauma and *C* and *D* are post-trauma angiograms. Numerals refer to seconds after injection. Note that in *D*, the peripheral cerebral arteries are still filled with contrast material 11 seconds after injection, while the angiographic appearance of the arteries in *A* and *C* is similar.

M-6 and 1.5 cc. in all others). Although the large volume of contrast material used cannot be entirely eliminated as a causative factor in the spasm seen, control injections of 3 cc. given 4 times in succession (M-9, Table I) failed to produce such spasm. Also, monkey M-6, which received 4 cc. per injection, showed no change in the caliber of the vessels. The injection time in this animal was so long that the circulation time could not be accurately estimated.

In 3 animals (M-22, 23 and C-1), intra-arterial blood pressure recordings were made before and after angiography and after trauma. The blood pressure recordings were equally normal at the time of the control and post-trauma angiography, even though one of the monkeys in which this was measured was accelerated to 3,400 g. when concussion was produced (M-22). Thus, the cerebral circulatory slowing observed angiographically was not due to hypotension.

DISCUSSION

The method of cerebral angiography, with carotid ligation and cannulation, provided a reproducible method for visualizing the cerebral vasculature. While the blood flow characteristics to a hemisphere are altered by ligation, it has been shown in man that the pressures in the intracranial vessels, after cervical carotid ligation, are about 60–80 per cent of the pre-ligation pressures.[1] Further, our control experiment showed that carotid ligation did not by

itself cause a breakdown of the blood-brain barrier. Although hypaque 50 per cent did cause a breakdown in the blood-brain barrier, it did not cause any significant change in the cerebral circulation time or appearance of the cerebral vessels angiographically. The slowing of the cerebral circulation seen in the interval 15 to 30 minutes following cranial trauma was thus, in fact, due to the trauma. The fact that the electrocardiographic findings and blood pressures were very nearly the same at the time of post-traumatic angiography as before the trauma excluded central cardiovascular causes, such as hypotension, for the cerebral circulatory slowing observed. It is interesting to note that the greatest degree of cerebral circulatory slowing was seen in the animal which was accelerated the most (M-22; 3,400 g.) (Fig. 4, *A–D*), while the circulatory slowing in an animal which was not concussed (M-11) was minimal. This implies some correlation between the severity of the trauma and the degree of circulatory slowing.

The actual relationship of circulatory slowing in the interval 15–30 minutes following trauma to the development of concussion is uncertain. In all of the animals in which the circulation time was measured angiographically before and after trauma, post-traumatic slowing was observed, even though one of these animals was not concussed. Since concussion in our experiments was a transient event which was generally well over by 15 minutes,[8] the circulatory studies which are to be done during and immediately following head trauma will be of more help in clarifying this possible relationship. It is, for instance, possible that the circulatory slowing is maximum initially and decreases thereafter.

Although the causes for the cerebral circulatory slowing in the early post-traumatic period are still obscure, it may be helpful to discuss the possible mechanisms. Perhaps it is simply secondary to arterial spasm. Spasm has been well documented following other insults to the brain such as rupture of aneurysms and, experimentally, following mechanical stimulation.[9] It has been shown that stimulation of the brain stem can alter cerebral blood flow, independent of cortical electrical activity, and

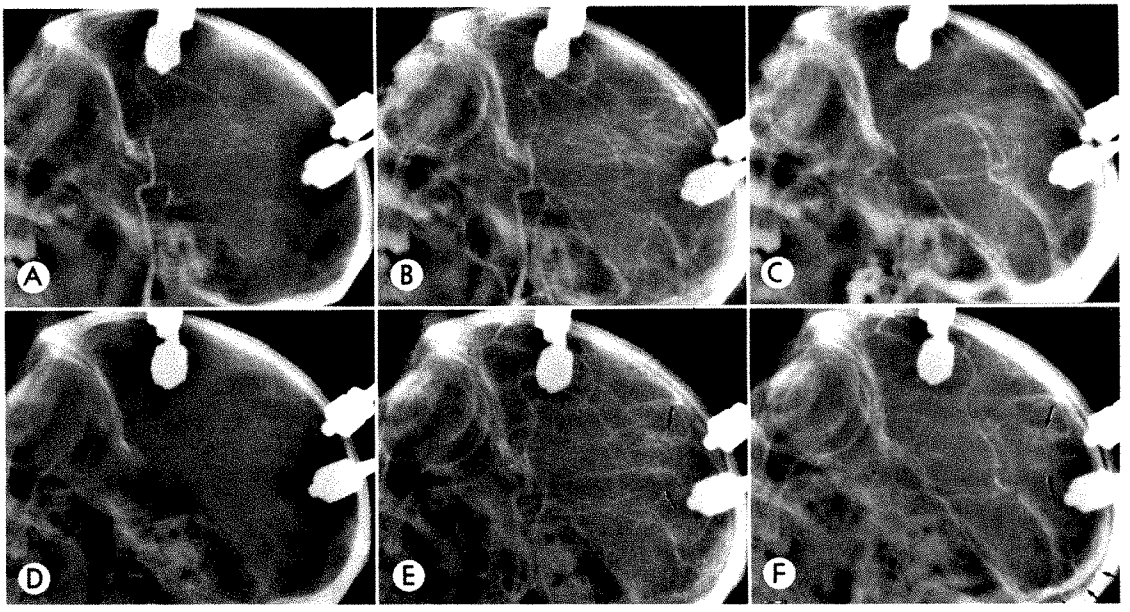


Fig. 5. Vascular stain and epidural hematoma after trauma (M-6, Table II). *A*, *B* and *C* are pre-trauma and *D*, *E* and *F* are post-trauma angiograms. Note the parietal stain (parietal arrows in *E* and *F*) after trauma. The first post-trauma angiogram (*D*) demonstrates that the stain is not artefactual. Note post-traumatic displacement of sagittal sinus at site of epidural hematoma (occipital arrows in *F*).

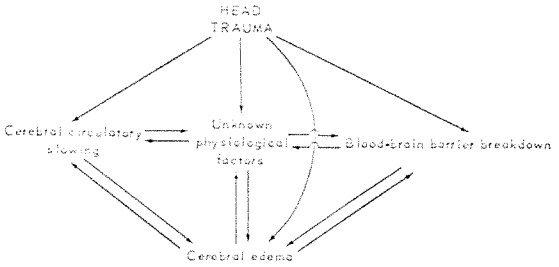


FIG. 6. Speculative relationships of primary cerebrovascular changes following head trauma.

occasionally this stimulation results in vasoconstriction.[5] Perhaps the brain stem damage demonstrated in the concussed animals in our experiments was, at least partly, related to the cerebral circulatory slowing observed. Very recently, Huber[4] pointed out that areas of arterial narrowing occur on cerebral angiograms of head trauma patients. If arterial spasm is causing the circulatory slowing seen in our experiments, then it must either be of a relatively minor degree or occur at the arteriolar level, since no consistent change in the caliber of the arteries or degree of peripheral vascular filling was noted when comparing pre- and post-trauma roentgenograms. It is recognized, however, that small changes in diameter, which might not be detected angiographically, could cause relatively large changes in the cross sectional area of a vessel and thus affect the vascular resistance.

Another possible explanation for the circulatory slowing observed is the development of cerebral edema. It has been shown that the volume of the brain increases after concussion as much as 3 to 3.4 per cent in the interval 16 to 26 minutes after the trauma.[10] Since the post-trauma angiograms in our experiments were made 15 to 30 minutes after the trauma, the relationship of circulatory slowing to cerebral edema may be significant. Even if related, however, it still remains to be shown whether circulatory slowing is due to the cerebral edema, or is an additional cause of it.

A third possibility for the cerebral circulatory slowing is the development of intravascular aggregations of blood cells.[2,7] These have been generally described, however, as following quite severe crushing injuries. Since circulatory slowing was observed following even relatively minor head trauma, the possibility of intravascular thrombi being the sole cause for the slowing is doubted.

The use of sodium fluorescein to study the breakdown of the blood-brain barrier after trauma provides a sensitive method which indicates the immediate location of at least one component of the vascular lesion. Since breakdown of the blood-brain barrier is usually associated with functional changes in nervous tissues,[3] it may well be that our observations of circulatory slowing in the angiogram is in some way related to the breakdown of the barrier, one basic expression of which may be cerebral edema.[6]

There are thus three cerebrovascular aspects of head trauma whose relationships must be clarified: slowing of the cerebral circulation, breakdown of the blood-brain barrier, and the development of cerebral edema. As illustrated in Figure 6, it may well be that they are related by a common factor, herein called "unknown physiologic changes." Such changes may be in the form of morphologic alterations in the glial barrier leading to breakdown of the blood-brain barrier, or to a biochemical alteration in the neural or glial elements which produces the same effects. Whether the cerebral edema which follows head trauma arises secondary to the "unknown physiologic changes" or to primary changes in the blood-brain barrier, a possible resultant could be cerebral circulatory slowing. Such slowing could then, in turn, aggravate the primary changes and lead to further trauma to the nervous system. However, it is quite possible that the circulatory slowing may be a primary change itself, in direct response to the trauma, which could then lead to cerebral edema or to breakdown of the blood-brain barrier, by way of the "unknown physiologic changes." In

order to help elucidate the sequences and nature of these time related events, further experiments are in progress.

SUMMARY AND CONCLUSIONS

The results of a series of experiments investigating the cerebral angiographic changes which take place in the interval 15 to 30 minutes following head trauma to primates are presented. Consistent cerebral vascular slowing is demonstrated angiographically. The possible mechanisms of this slowing and its relationship to the degree of trauma, to the production of concussion and to the blood-brain barrier breakdown observed are discussed.

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NONPATHOLOGIC AND PATHOLOGIC RELATIONSHIPS BETWEEN THE LOWER CERVICAL VERTEBRAE*

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PERUSAL of the literature reveals a diversity of opinions on the borderline between normal and pathologic cervical vertebral realtionships. This is particularly true for minor displacements, which are interpreted as subluxations by some authors, while others believe that they should be regarded as physiologic. The lack of agreement must be partly ascribed to an insufficient knowledge of the normal intervertebral motor mechanism, and to the resulting lack of familiarity with the relative positions of the vertebrae in unusual postures of the spine. Normally, roentgenographic examination of the cervical spine demonstrates the vertebrae in standard positions, and information is therefore obtained only on the vertebral relationships in these positions.

The purpose of this paper is to study the mechanism of normal motion in various unusual postures of the cervical spine and thereby establish criteria for a more accurate differentiation between pathologic and nonpathologic vertebral relationships. The discussion is confined to the lower cervical region, from C2 to D1, which widely differs functionally from the upper cervical region with its atlanto-occipital and atlanto-axial articulations. The lower cervical region includes 6 mobile intervertebral connections, the so-called motor segments (Junghanns) C2/C3 through C7/D1. Movement in these segments is chiefly in the sagittal plane (flexion, extension). To study this movement, roentgenograms of the cervical spine are obtained in maximal flexion, in neutral position and in maximal extension. The most reliable results are secured with the subject in a sitting position (Fig. 1, *A–C*).

In the literature[1,4] the roentgenologic studies of movement in the sagittal plane have been chiefly quantitative, measuring the excursions between the vertebrae; the qualitative features—the mechanism of movement—have been neglected, although anatomists[2,3,7,20,21] were interested in analysing the mechanism of movement even before the roentgen era. Fick[7] was familiar with the position of the motor axes in the cervical region, but the data collected by him received hardly any attention in the roentgenologic literature.

NONPATHOLOGIC RELATIONSHIPS

MOTOR DIAGRAMS

For an analysis of the mechanism of movement of the lower cervical vertebrae, the lateral roentgenograms obtained in maximal flexion and maximal extension are combined to form the so-called motor diagrams, ("Bewegungsdiagramme," Buetti-Bäuml[4]), as shown in Figure 2. Type I shows the superior vertebra in a fixed position of maximal flexion and maximal extension. Type II depicts the same movement, but with the inferior vertebra in a fixed position. A typical feature of the mechanism of movement of the lower cervical vertebrae is the sliding motion of the superior vertebra relative to its inferior partner. In consequence of this sliding motion, a step ("step-off", Hadley[10]) is formed along the ventral wall of the spinal canal in flexion, and a reversed step in extension. In flexion, the dorsal borders of the vertebral bodies are thus arranged like a flight of steps, while in extension the arrangement resembles a washboard pat-

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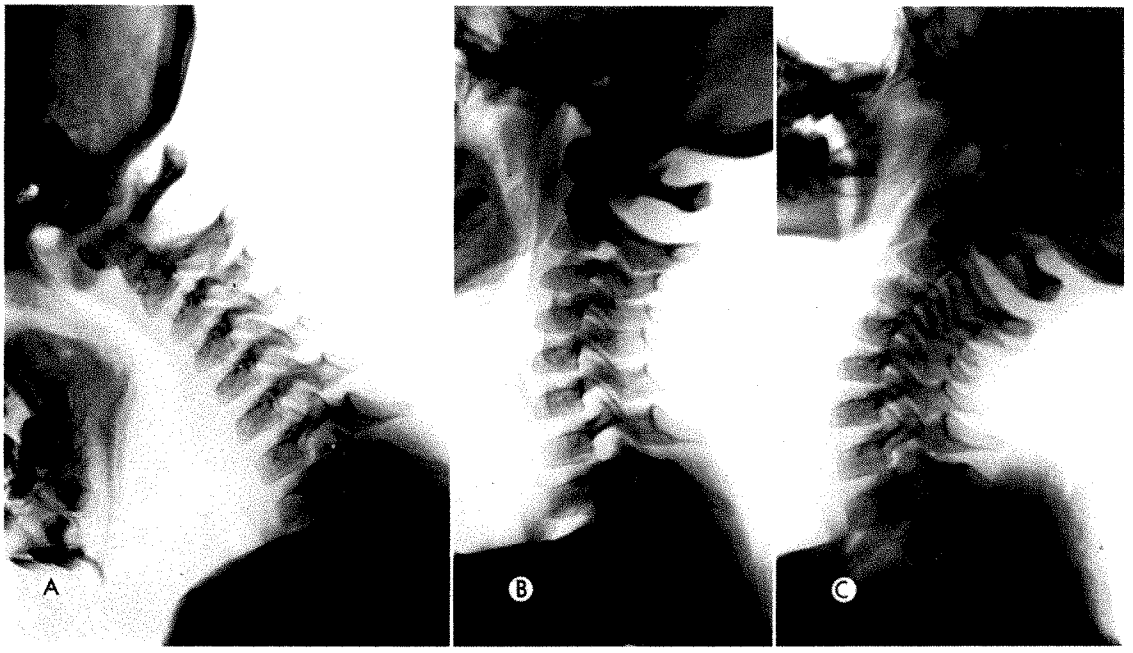


Fig. 1. Lateral roentgenograms of the cervical spine of a 5 year old boy in (*A*) flexion, (*B*) neutral position, and (*C*) extension.

tern. The extent of the sliding movement is dependent on the extent of the excursion and on the position of the motor axis.

Extent of Normal Excursion

The extent of normal excursion in the lower cervical region has been determined by various investigators[1,4,13,18]; it is presented in Table 1. These values apply to young adults. In children the excursions observed in segment C_2/C_3 are considerably greater; in 6 normal children under 16 years

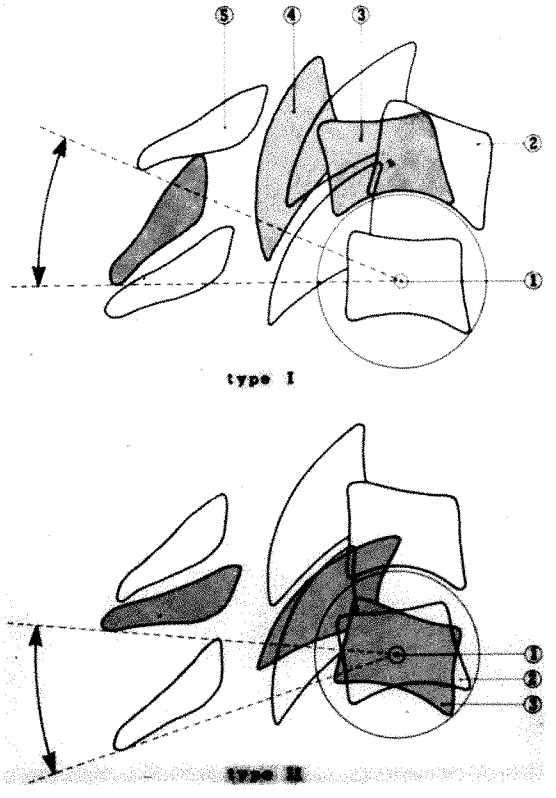


Fig. 2. Diagrams of two types of motor function indicating the relative movement in the sagittal plane of a pair of lower cervical vertebrae. In type I, the superior vertebra is supposed to be fixed, while the inferior vertebra is in the extreme flexion and extension positions. In type II the reverse is presumed. (1) Motor axis; (2) flexed position of the mobile vertebra; (3) extended position; (4) articular process; (5) spinous process. Total movement range 29°. (Reproduced with permission from *J. Bone & Joint Surgery*, 1963, 45-*A*, 1671–1678).

TABLE I

AVERAGE EXCURSIONS AND RANGE OF VARIATION OF THESE EXCURSIONS OF THE LOWER CERVICAL MOTOR SEGMENTS IN THE SAGITTAL PLANE, ROENTGENOLOGICALLY DETERMINED IN YOUNG ADULTS

	Bakke[1]	de Sèze *et al.*[18]	Buetti-Bäuml[4]		Penning	
C2/C3	12.6°	13°	10½°	5–18°	12½°	(5) 8–16°
C3/C4	15.4°	15½°	17°	13–23°	18°	13–26°
C4/C5	15.1°	19°	21°	16–18°	20°	15–29°
C5/C6	20.4°	27½°	22½°	18–28°	21½°	16–29°
C6/C7	17.0°	17½°	18°	13–25°	15½°	(6) 12–25°
C7/D1	10.2°	12½°	—	— —	10°	8–12°
	average excursion	average excursion	average excursion	range of variation	average excursion	range of variation

of age, we found an average excursion of 20° (14–24°).

AXIS MOVEMENT

The position of the axis movement was studied in 30 vertebral columns; it was always found in the dorsal half or in the central part of the inferior vertebral body. The axes of the superior motor segments are in a position more caudal and dorsal than that of the axes of the inferior motor segments. The axis of C6/C7 lies in or just below the center of the cranial roofplate of C7. The average position of the motor axes is shown in Figure 3.

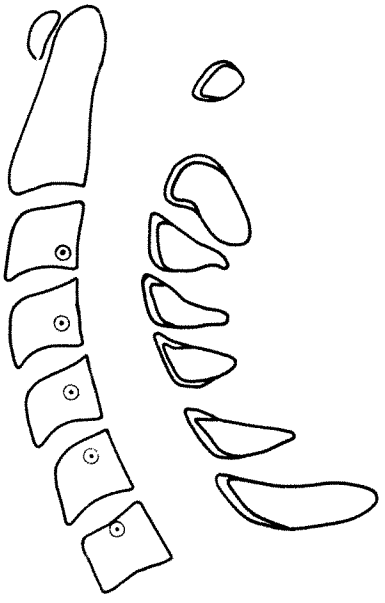


FIG. 3. Average position of the axes of movement (sagittal plane) of the lower cervical vertebrae in 30 normal test subjects.

PSEUDO-SUBLUXATION

The above findings indicate that, excursions in all lower cervical motor segments being equal, step formation will be most pronounced in segment C2/C3. In adults, however, step formation shows the same degree throughout because of less marked mobility in the upper cervical region. Children may show striking step formation in segments C2/C3 and C3/C4, particularly in flexion of the cervical spine (Fig. 1). In the past, this has repeatedly led to a diagnosis of subluxation; today this condition is known as pseudo-subluxation.[6,11] Of course, the step formation is characteristic for the flexed cervical spine and not for the cervical spine in a neutral position. However, the investigator should not be misled by the position of the head when estimating the position of the lower cervical spine. In recumbent patients (particularly when they are unconscious) the cervical spine will sag and thus assume flexed position, while the head is in the neutral position. Step formation in the segments C2/C3 and C3/C4 is normal in this case.

ARCUAL KYPHOSIS

A similar phenomenon can be observed in some cases of so-called "stiff neck" in young subjects, where an acute arcual

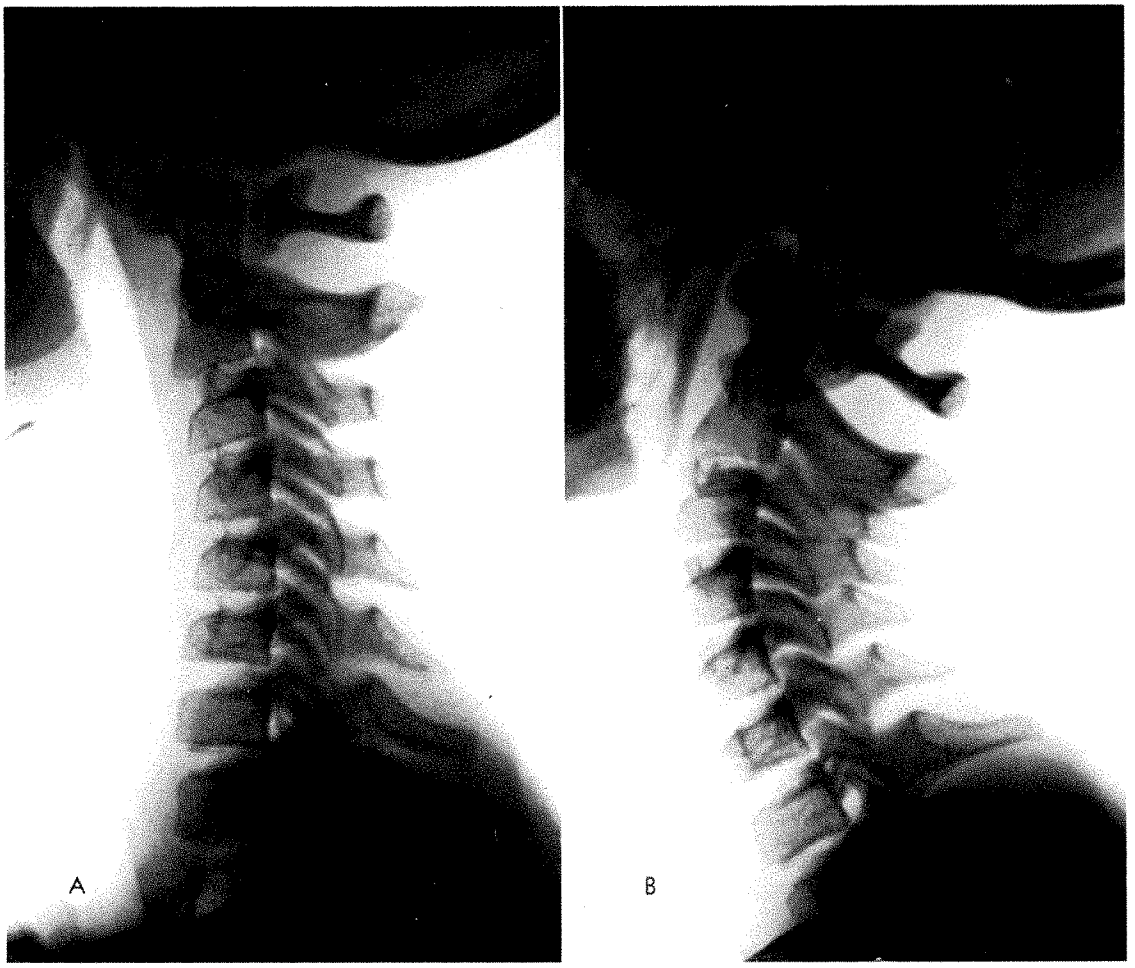


Fig. 4. (*A*) Lateral roentgenogram of the cervical spine of an 11 year old boy who developed nuchal rigidity and pain following a playful bout of wrestling. There is arcual kyphosis. The patient is sitting with the head in ortho-optic position. (*B*) Roentgenogram 1 week later. The patient is again sitting with the head in ortho-optic position. The normal lordosis is restored. There were no symptoms.

kyphosis, as the result of a reflex spasm of the paraspinal muscle groups, follows trivial injury of the neck or infection of the upper respiratory tract[6] (Fig. 4, *A* and *B*). Once, we encountered the combination of an arcual kyphosis with a prevertebral hematoma (Fig. 5).

ARCUAL SCOLIOSIS

In addition to flexion and extension, the lower cervical motor segments allow a rotation which amounts to some 6–8° to the right and to the left for each segment.[13] Because of the oblique position of the intervertebral joints, rotation is always combined with lateral flexion, and *vice versa*. If the cervical spine with the head is laterally flexed, while the head is not rotated, the rotation of the lower cervical spine is completely compensated by a counter rotation in the atlanto-axial joint.[14] An anteroposterior roentgenogram obtained in this posture reveals a deviation of the spinous process of the axis, contralateral to the side of flexion of the head. A similar appearance is observed in the scoliosis of the cervical spine which can be compared to a cervical spine in lateral flexion. Thus in scoliosis deviation of the spinous process of the axis is a physiologic phenomenon. The direction

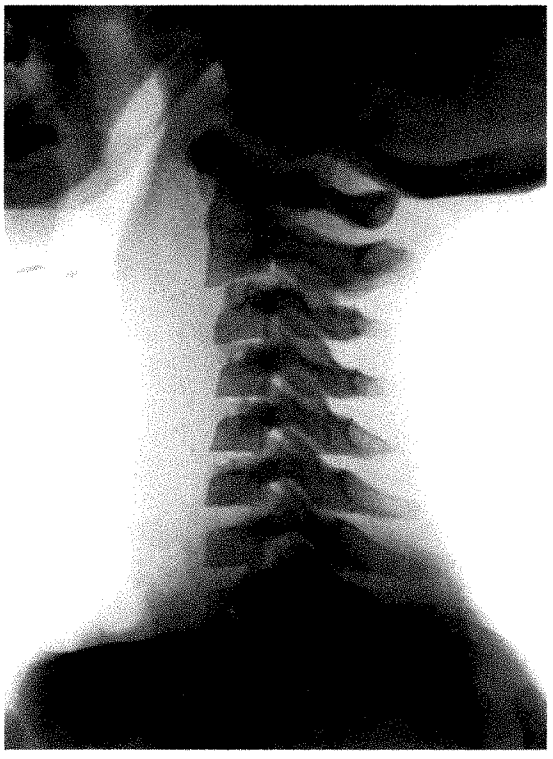


FIG. 5. Lateral roentgenogram of the cervical spine of a 4 year old girl, who was overthrown by a cyclist. There is a prevertebral hematoma (increased distance between the windpipe and the vertebral column). There is arcual kyphosis of the cervical spine.

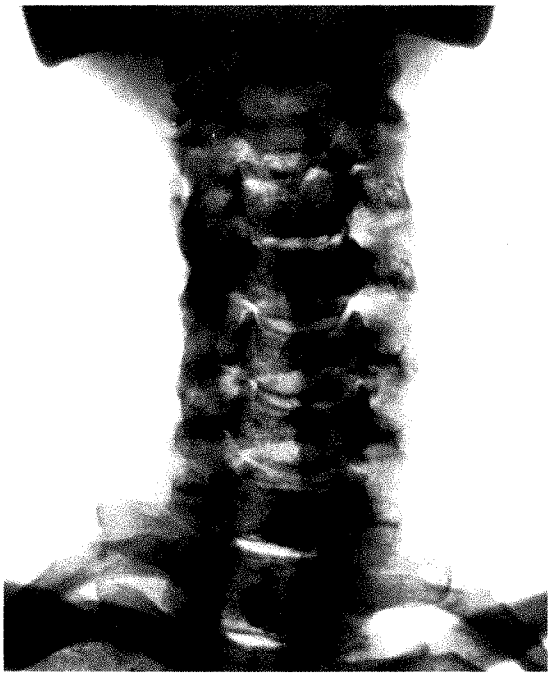


FIG. 6. Frontal roentgenogram of a scoliotic cervical spine with the convexity to the left. The deviation of the spinous process of the axis to the left must be regarded as a physiologically concomitant phenomenon.



FIG. 7. (*A*) Lateral roentgenogram of the cervical spine of an 11 year old boy after injury, while diving head first. The roentgenogram was taken in the sitting position. There is isolated flexion of segment C_2/C_3, with kyphotic angulation of the spine, due to tearing of the posterior ligaments. There is a chip fracture of the spinous process of the axis. (*B*) Line drawing of *A*.

of the deviation in these cases corresponds with the convexity of the scoliosis (Fig. 6).

PATHOLOGIC RELATIONSHIPS

ANGULATIONS

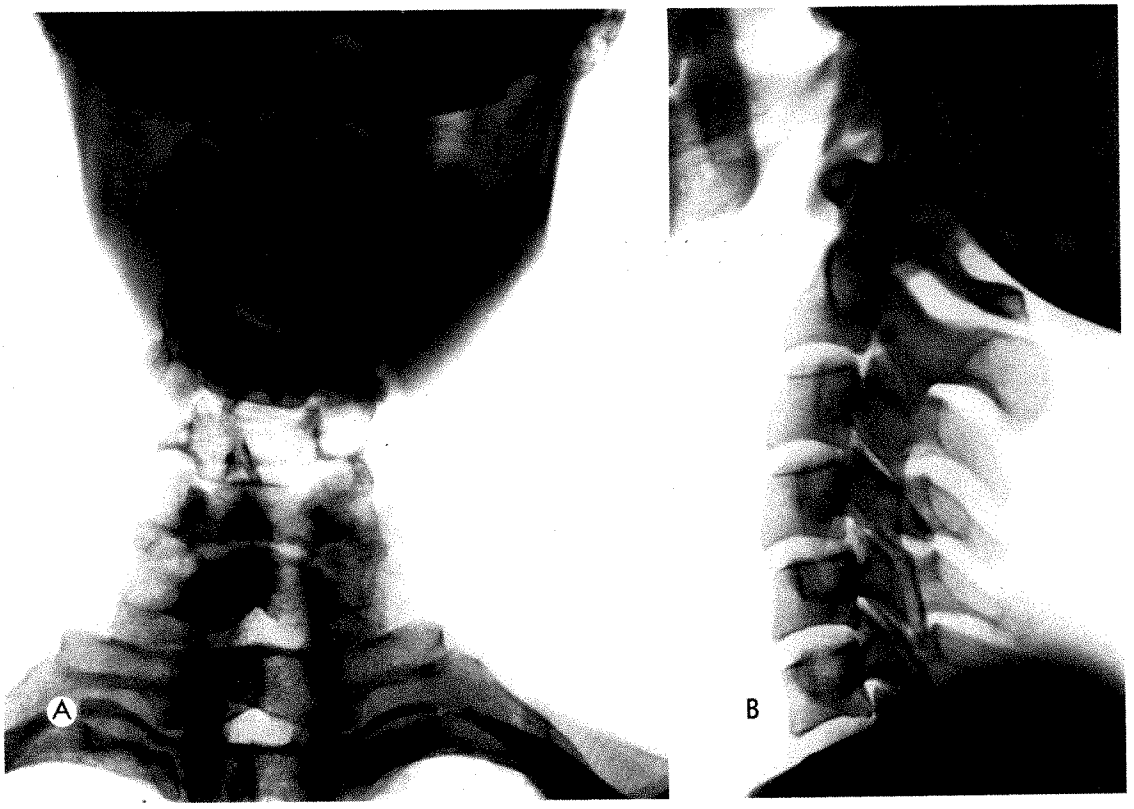
The so-called angulations ("Knickungen") are foremost among the pathologic vertebral relationships.[4,10] In normal subjects the cervical spine is never angulated, but always shows smooth curves. Strictly



← Fig. 7.

speaking, the relationship between angulated vertebrae as such is not pathologic because the vertebrae assume a position that is functionally possible; however, the position assumed is not in accordance with the over-all picture. An example of a *kyphotic angulation* is shown in Figure 7, *A* and *B*, where the spine as a whole is in a neutral position, while motor segment C_2/C_3 shows maximal flexion. The term subluxation is not justified by the step formation between the two vertebrae nor

Fig. 8. (*A*) Anteroposterior roentgenogram of the cervical spine in a case of traumatic compression of the left articular process of C_4. There is scoliotic angulation in the segment C_4/C_5. (*B*) Lateral roentgenogram shows the compressed articular process of C_4 to greater advantage. According to Whitley and Forsyth[25] the inferior facet tends to be horizontal and the lateral mass is almost triangular in shape. At the same time there is a slight anterolisthesis of C_4/C_5.



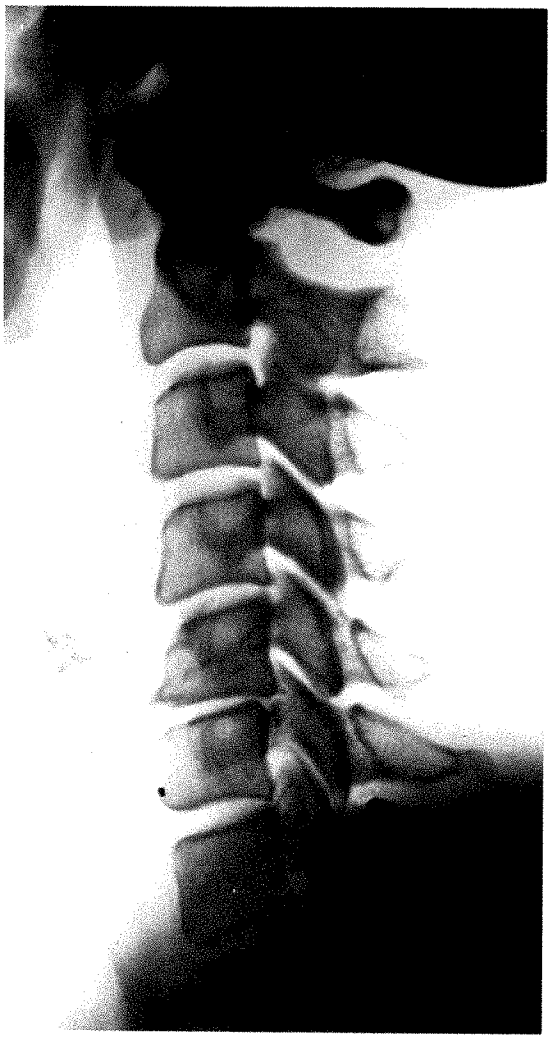


FIG. 9. Retrolisthesis of C_5/C_6 in a 50 year old woman caused by narrowing of the intervertebral disk space.

by the diastasis of the spinous processes, because these are physiologic phenomena in flexion. The cause of the kyphotic angulation is traumatic hyperflexion of the neck associated with rupture of the posterior ligaments at this level (flexion sprain). Minor kyphotic angulations may result from disk degeneration. The reverse of a kyphotic angulation is a *lordotic angulation* (or isolated flexion). As has been pointed out isolated rotation of one of the lower cervical motor segments is of necessity associated with isolated lateral flexion, visible on anteroposterior roentgenograms. We propose to refer to this condition as *scoliotic angulation*. An example is presented in Figure 8, *A* and *B*.

SLIPPING (OLISTHESIS)

Luxations or dislocations can be defined as standing disruptions of the physiologic contact between articular surfaces.[5] Strictly speaking, dislocations of vertebral bodies are impossible because the bodies are connected not by true joints, but by disks.

Retrolisthesis. The vertebral relationship, demonstrated in Figure 9 may therefore better not be described as a dislocation or (sub) luxation because there is no complete or partial disruption of the contact between the articular surfaces (of the intervertebral joints). We prefer to refer to this condition as retrolisthesis.[8] Retrolisthesis (from the Greek word olisthanein = to slip) is a not infrequent finding in elderly cevical spines with degenerated disks, particularly in the C_3/C_4 and C_4/C_5 segments. The sliding seems to be a logical sequela of the oblique position of the intervertebral joint surfaces as a result of which the superior vertebra approaches its inferior partner in dorsocaudal direction due to narrowing of the intervertebral disk. However, narrowing of the disk and retrolisthesis are not always concomitant; the cause therefore remains somewhat obscure. Retrolisthesis may not be interpreted as the result of an acute trauma to the neck. It derives its significance from its property to narrow the anteroposterior diameter of the spinal canal and may contribute to the pathogenesis of the so-called spondylotic myelopathy and of medullary lesions in hyperextension traumata of the cervical spine.[15]

Anterolisthesis. The reverse of retrolisthesis is anterolisthesis; *i.e.* forward slipping of a vertebra upon its inferior partner without disruption of the contact between the articular surfaces (Fig. 8, *A* and *B*; and 10, *A* and *B*). It is usually caused by atrophy of the cartilage of the intervertebral joints as the result of degenerative

(*Text continued on page 1047*)

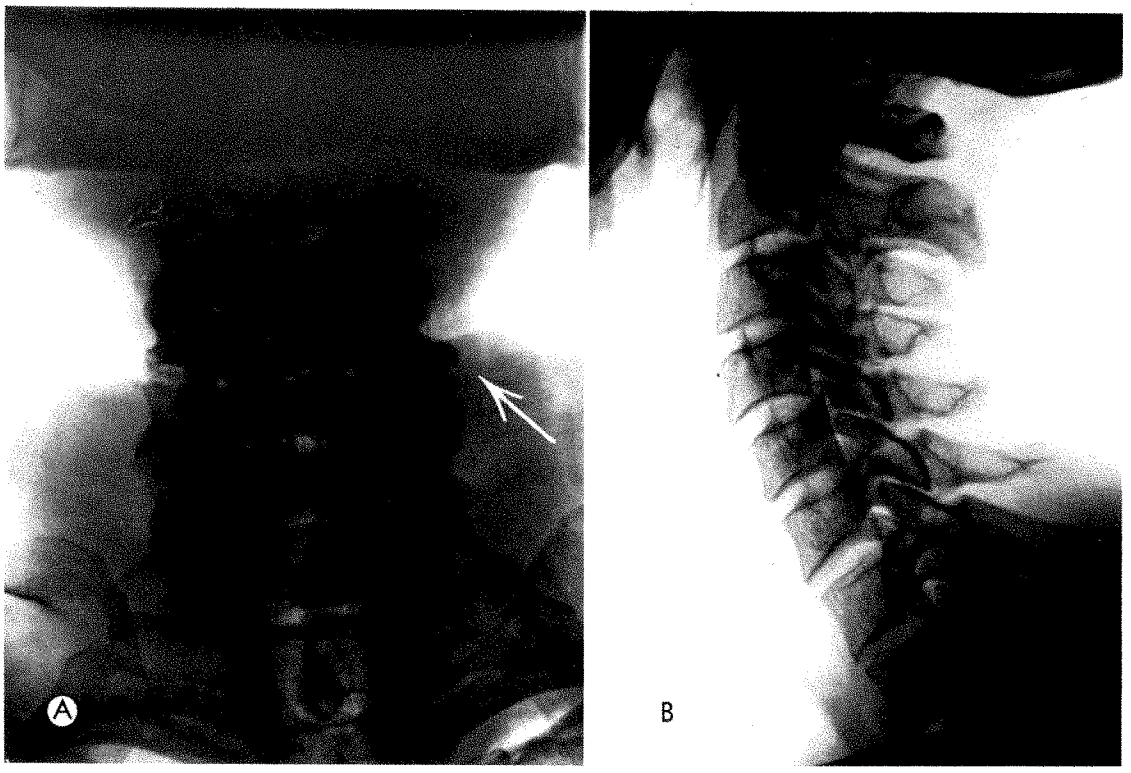
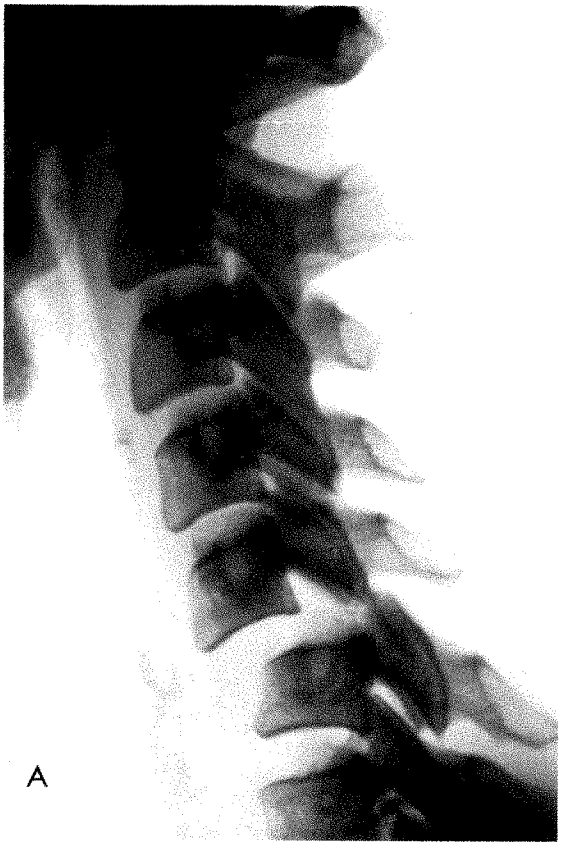


FIG. 10. (*A* and *B*) Anterolisthesis of C_4/C_5 caused by arthrosis deformans of the intervertebral joints (arrow).



FIG. 11. Spondylolisthesis of $C6$/C_7 with a bilateral defect in the interarticular portion of the arch of C6. This anomaly was an accidental finding in a 40 year old clerk, who for 3 years complained of some difficulty in swallowing and slight pain in the jugular area. The larynx, pharynx and esophagus proved to be normal.



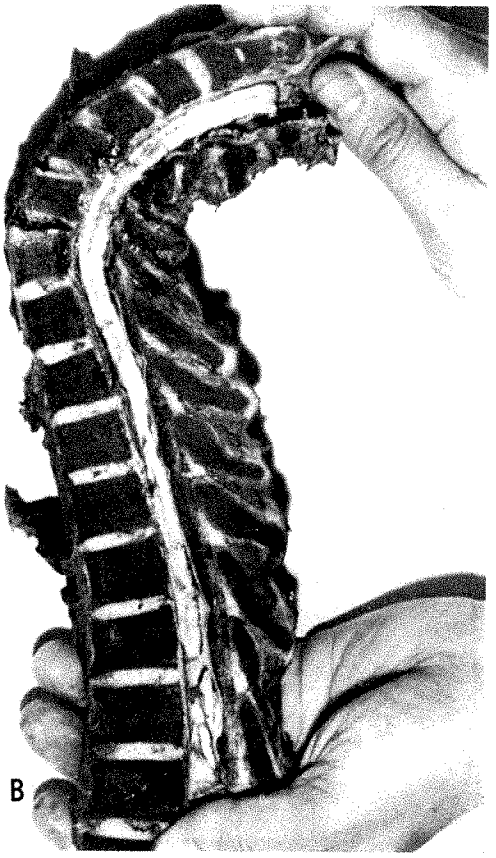
A



B



A



B

Fig. 12. (*A*) Flexion luxation and bilateral interlocking of the articular processes of $C_5/C6$ in a male, 42 years of age, with quadriplegia. Reduction by means of skull traction remained unsuccessful. (*B*) Roentgenogram 6 months after open reduction and bone grafting. A slight anterolisthesis of $C_5/C6$ persists. There is slight bony bridging along the anterior longitudinal ligament. The patient made a partial recovery from the quadriplegia.

Table II

VARIOUS TYPES OF PATHOLOGIC RELATIONSHIPS BETWEEN THE VERTEBRAE IN THE LOWER CERVICAL REGION (C_2–D_1), EXCLUDING FRACTURE DISLOCATIONS

Angulation	(a) kyphotic (isolated flexion)
	(b) lordotic (isolated extension)
	(c) scoliotic (isolated rotation)
Slipping (olisthesis)	(a) retrolisthesis
	(b) anterolisthesis
	(c) spondylolisthesis
Dislocation (luxation)	(a) flexion luxation
	(b) hyperflexion (without interlocking of articular processes)
	(c) rotation luxation

Fig. 13. (*A*) Lateral roentgenogram of the cervical spine of a 50 year old male who fell downstairs on his face and immediately showed signs of a complete quadriplegia. Roentgenographically the cervical spine appears unimpaired. (*B*) Postmortem specimen of the spine shows laceration of the cervical cord at the C_4–C6 level. On forced hyperextension the complete disruption of the anterior longitudinal ligament and of the intervertebral disk of $C_5/C6$ and $C6/C_7$ with compression of the spinal cord (Taylor-mechanism) is clearly seen.

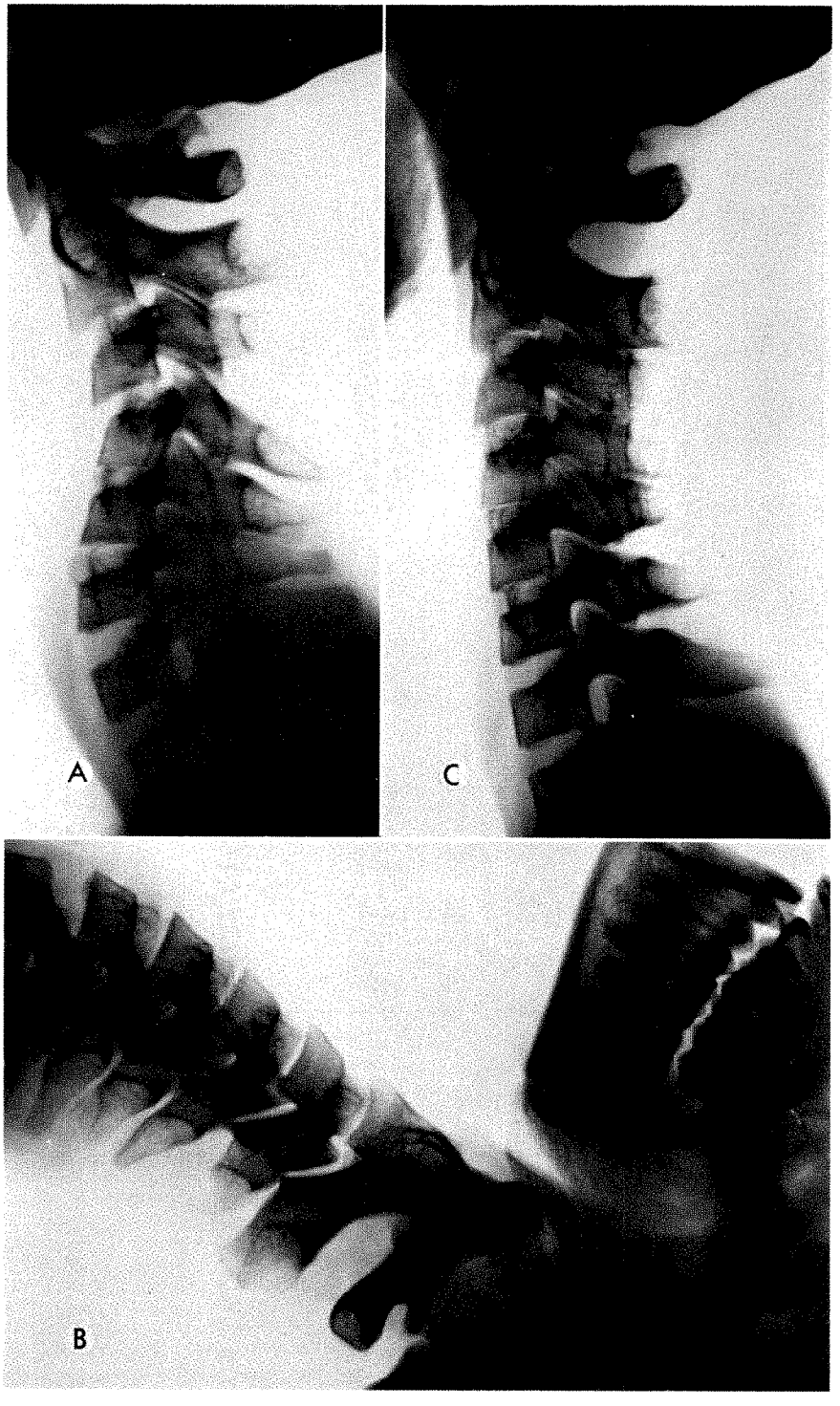

A
C
B

Fig. 14. (*A*) Kyphotic hyperangulation of segment C_3/C_4 in a boy 14 years of age, after a diving accident. No interlocking of the articular processes is present. (*B*) On passive extension of the unsupported head, the normal curvature of the spine is reestablished—a fact which in case of interlocking of the articular processes would be impossible. (Roentgenogram taken with the patient on his back, head and neck in retroflexion.) (*C*) Restoration of the normal lordosis following posterior spinal fusion of C_2–C_5.

(Junghanns' "pseudospondylolisthesis"[17]) or rheumatoid arthritic[9,12,19] changes.

Spondylolisthesis. This is a special form of anterolisthesis, which is common in the lumbar region but rare in the cervical spine. The first case of spondylolisthesis was described by Pearlman and Hawes.[16] In spondylolisthesis, too, there is no luxation because the contact between the articular facets has remained intact; the slipping is caused by a bilateral defect in the vertebral arch (Fig. 11). Traumatic anterolisthesis was seen by us as the result of compression of the articular processes (Fig. 8, *A* and *B*).

Dislocation or Luxation

True dislocation or luxation exists in the event of an interlocking of the articular processes. If it is unilateral, it can be referred to as a *rotation luxation*, while the term *flexion luxation* is appropriate if it is bilateral (Fig. 12, *A* and *B*). Roentgenologically the two types are best differentiated by means of roentgenograms in the oblique Buetti-Bäuml projections.[4] *Extension luxations* are not encountered because the requirements for interlocking of the articular processes are lacking. Traumatic hyperextension of the cervical spine may result in a serious laceration (sprain) of a motor segment, even with compression of the cervical cord, but directly after the trauma the cervical spine resumes its normal position so that the lesion is not demonstrable roentgenographically (Taylor-mechanism)[23,24,25] (Fig. 13, *A* and *B*). Traumatic hyperflexion with serious laceration of a lower cervical motor segment results in a hyperflexion position of the severed segment (Fig. 14, *A*–*C*). Strictly speaking, the term flexion luxation could be used in these cases because such extreme flexion is impossible under physiologic conditions. On the other hand, there is no interlocking of articular processes; on extension the spine resumes its normal curve. We propose the term *kyphotic hyperangulation* for these cases.

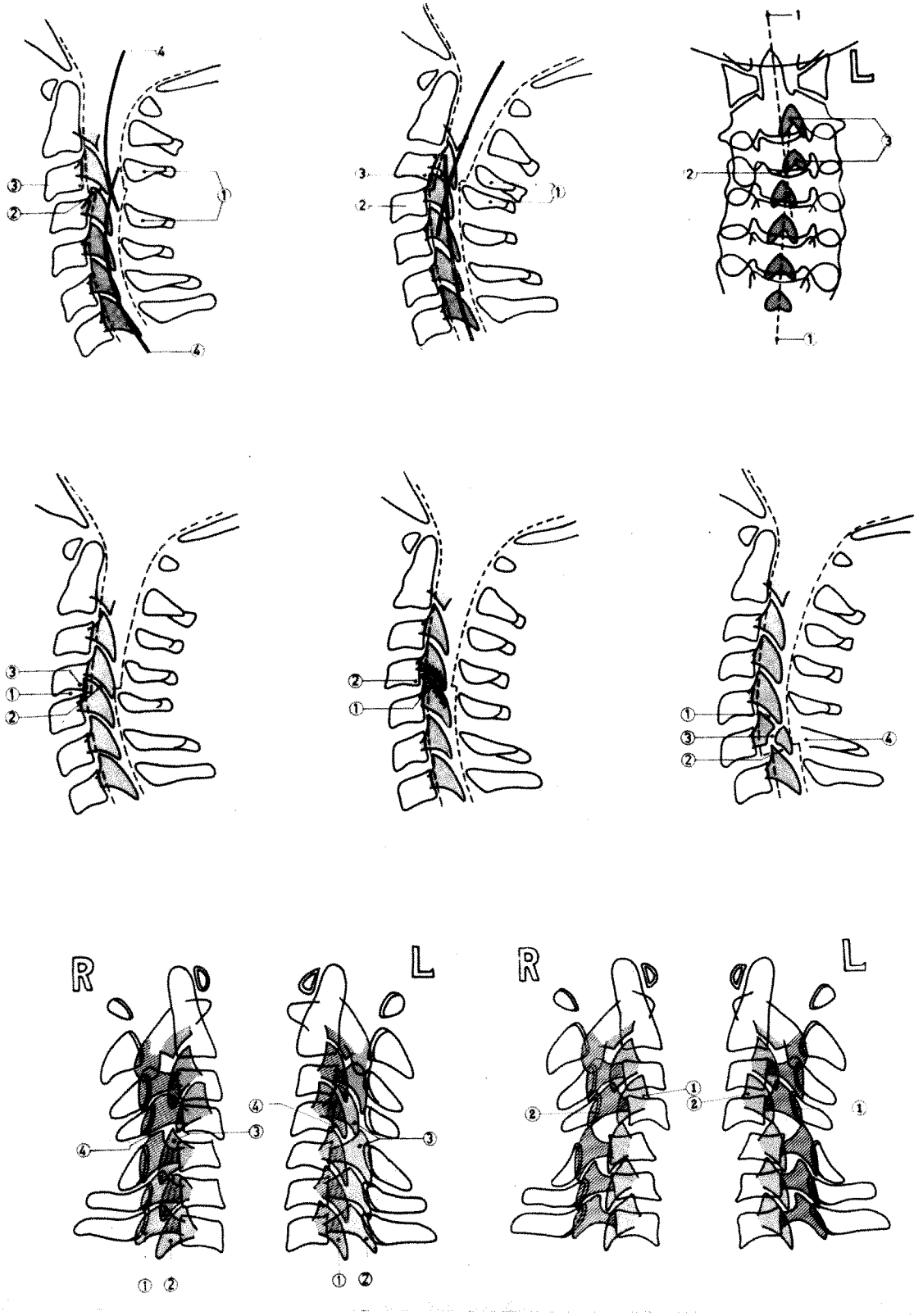

R
L
R
L

Fig. 15. Schematic views of the different kinds of pathologic relationships between the lower cervical vertebrae.

Upper row, *left:* kyphotic angulation of C_3/C_4: (1) spinous processes, (2) intervertebral joint, (3) step formation between vertebral bodies of C_3 and C_4, (4) axis of spinal canal; *center:* lordotic angulation of C_3/C_4: (1) spinous processes, (2) vertebral body, (3) step formation between bodies of C_3 and C_4; *right:* scoliotic angulation of C_3/C_4: (1) axis of spinal canal, (2) severed motor segment, (3) shift of spinous processes.

Middle row, *left:* retrolisthesis of C_4/C_5: (1) narrowed disk space, (2) normal relationship of intervertebral joint, (3) retrolisthetic vertebral body; *center:* anterolisthesis: (1) narrowed intervertebral joint space, (2) anterolisthetic intervertebral body; *right:* spondylolisthesis of $C6/C_7$: (1) normal intervertebral joint of $C_5/C6$, (2) normal intervertebral joint of $C6/C_7$, (3) defect in the interarticular portion of the articular processes of C6.

Lower row, *left:* right and left oblique projections of Buetti-Bäuml in the case of an unilateral interlocking of the articular processes of C_4/C_5 (rotation luxation): (1) articular processes of right side, (2) articular processes of left side, (3) site of interlocking (left), (4) no interlocking on right side; *right:* right and left oblique projections of Buetti-Bäuml in the case of bilateral interlocking of the articular processes of C_4/C_5 (flexion luxation): (1) interlocked articular process of C_4 on left side, (2) interlocked articular process of C_4 on right side.

SCHEMA OF PATHOLOGIC RELATIONSHIPS

The various forms of pathologic relationship between the (otherwise unsevered) lower cervical vertebrae are listed in Table II, while schematic views are given in Figure 15. The term subluxation has been avoided advisedly, not only because it is ill-defined, but also because it is frequently misused and therefore more likely to cause confusion than to improve understanding.

SUMMARY

The knowledge of the normal mechanism of movement between the lower cervical positions in various extreme postures of the vertebral column, permits a more exact differentiation between pathologic and non-pathologic vertebral relationships. The various types of pathologic relationship (angulation, slipping and luxation) and their differential diagnosis are discussed in detail.

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CERVICAL DISK DISEASE SIMULATING INTRAMEDULLARY NEOPLASM BY MYELOGRAPHY*

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IT IS well known that cervical spondylosis or central disk herniations can produce neurologic changes which mimic those accompanying an intramedullary tumor of the cervical spinal cord. That myelographic deformities simulating an intramedullary tumor also can be produced by cervical disk disease has been reported only rarely. It is the purpose of the author to report 2 such cases. Both patients were somewhat younger than usual for degenerative disk disease, and in neither was there a history of trauma.

REPORT OF CASES

CASE I. A 34 year old female noted numbness of her fingers 1 month prior to admission, followed by symmetric numbness of both legs below the knees. With the latter, she noted that the right foot tended to drag with walking.

On neurologic examination, no abnormality of cranial nerve function was found. Sensation to light touch and pin prick was diminished on both palms and in both lower extremities below the knees. Position sense was intact. Deep tendon reflexes were generally hyperactive, with bilateral ankle clonus and bilateral Hoffmann reflexes. A questionable Babinski reflex was present on the left.

Nerve conduction time was tested in the right upper extremity, and was normal. Routine laboratory studies were within normal limits. A clinical diagnosis of multiple sclerosis and cervical cord tumor was entertained.

Roentgenograms of the cervical spine showed moderate narrowing at the interspace between the fourth and fifth cervical vertebrae, with anterior (but no definite posterior) spurring. The anteroposterior diameter of the spinal canal was 14 mm. at this level. Cervical myelography was performed. The fluoroscopic examination revealed abrupt widening of the cervical spinal cord, centered at the C_4–C_5 disk level and extending over a length of about 3.5 cm. (Fig. 1). On the roentgenogram made in lateral position, however, the cord appeared normal in size. There was a deep impression against the ventral surface of the contrast medium opposite the C_4–C_5 disk level (Fig. 2). A roentgenologic diagnosis of disk protrusion with cord compression was made.

The patient was transferred to the Neurosurgical Service. Under general anesthesia a bilateral laminectomy was performed on the third, fourth, and fifth cervical vertebrae. The cervical spinal cord at this level was displaced posteriorly by a large central disk protrusion; the disk was firm but did not seem to be calcified. The cervical cord appeared to entirely fill the compressed dural compartment. Four digitations of the dentate ligament were divided on each side, and the surgical incision was closed.

Following operation, the patient's gait became normal. Some sensory loss in the lower extremities was present at the time of her last clinic visit, 9 months after operation, but it was felt that her condition had improved.

CASE II. A 41 year old female was admitted to the North Carolina Memorial Hospital with a chief complaint of pain in her neck and arms. Several weeks prior to admission, the patient noted that the right arm seemed to tire more easily than usual. A sudden onset of pain in the right arm, radiating to the right shoulder and neck, had occurred 10 days before admission. Subsequently, there was pain in the left shoulder and left arm as well. On the day before admission she noted weakness of the right arm and leg, as well as inability to void.

General physical examination was not remarkable. Neurologic examination revealed no abnormality of cranial nerve function. There was a marked decrease in motor strength in the right upper and lower extremities, with absent deep tendon reflexes on the right. Tenderness was noted over the lower cervical spine.

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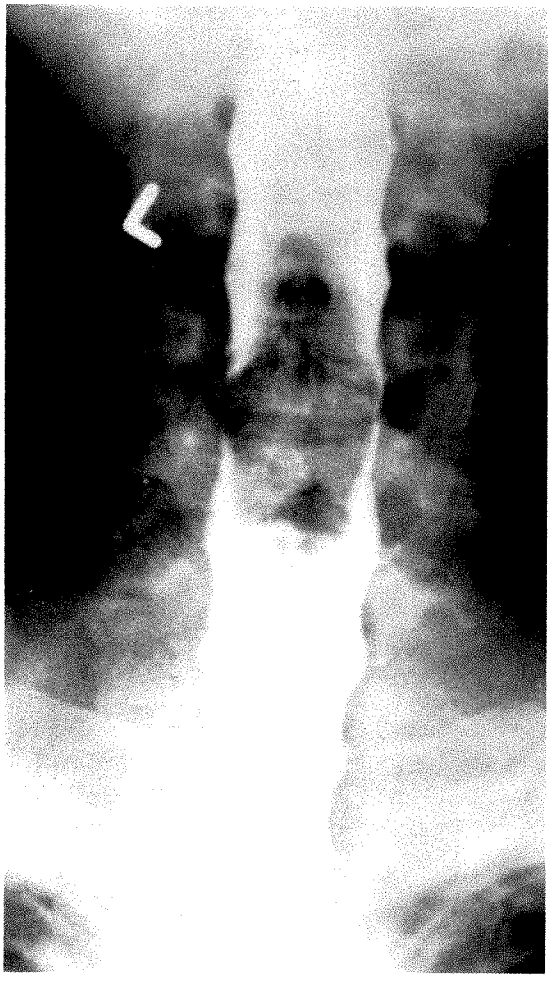


Fig. 1. Case 1. Frontal myelogram shows the cervical spinal cord to be abruptly widened at the level of the fourth and fifth cervical vertebrae.

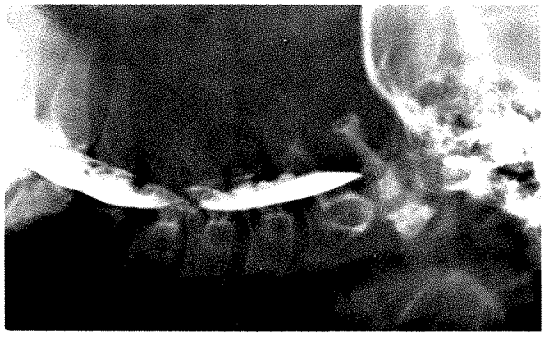


Fig. 2. Case 1. Myelogram made in lateral position shows no widening of the cervical cord in its anteroposterior dimension. A deep impression is evident in the ventral surface of the contrast column, opposite the disk space between the fourth and fifth cervical vertebrae.

Sensation of pin prick was diminished over the right upper extremity, although position sense was intact. No definite neurologic disturbance of the left extremities was found.

Routine laboratory studies were not remarkable.

Roentgenograms of the cervical spine showed moderate narrowing of the disk space between the fourth and fifth cervical vertebrae, with slight marginal spurring (Fig. 3). The anteroposterior diameter of the spinal canal was 15 mm. at C_4 and C_5.

Cervical myelography was performed. The cervical spinal cord appeared widened from C_4 to C6, over a distance of about 6 cm. (Fig. 4). In the small amount of contrast medium which passed around the apparently enlarged cord, some irregularity was noted on the right, opposite the C_5–C6 disk space. On the roentgenogram made in lateral position (not available

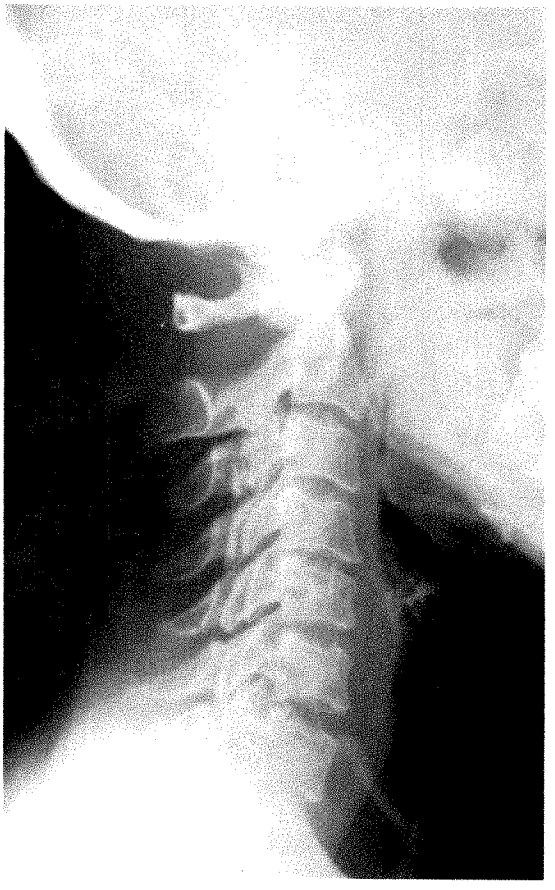


Fig. 3. Case II. Lateral roentgenogram shows slight narrowing of the disk space between the fourth and fifth cervical vertebrae.

for reproduction), there was slight indentation in the ventral margin of the contrast column at both the C_4–C_5 and C_5–C_6 disk levels. It was felt that, although an intramedullary tumor could not definitely be excluded, the findings seemed best explained by central disk herniations at C_4–C_5 and C_5–C_6.

A surgical laminectomy of the fourth, fifth, sixth and seventh cervical vertebrae was performed. The cervical spinal cord was widened and was displaced posteriorly. A midline myelotomy was done; there was no evidence of an intramedullary neoplasm. Firm, rigid bars extended posteriorly from the intervertebral spaces at C_4–C_5 and C_5–C_6. Four digitations of the dentate ligament were divided bilaterally, and the surgical incision was closed.

Muscle power in the right extremities returned to normal, although some disturbance of sensation persisted. Pain persisted intermittently in the right shoulder and arm.

DISCUSSION

Diverse neurologic changes may result when the cervical spinal cord is compressed by disk herniations or spondylosis. Clinical findings can be highly suggestive of multiple sclerosis,[1,7,10] amyotrophic lateral sclerosis,[5,10] foramen magnum tumors,[7] or cervical cord tumors.[1,3,5,7] Elevation of the spinal fluid protein level is not rare, even in the absence of a complete subarachnoid block.[4,7,10] The difficulties in clinical diagnosis may be further compounded by the absence of any symptoms in the neck[3,5] or even in the upper extremities.[1]

The differentiation of mass lesions affecting the cervical spinal cord usually can be made by myelography. Cramer and Hudson[2] have emphasized that, with lesions of the cervical intervertebral disks, the size of the spinal cord is contained within normal maximal limits as seen on myelograms. However, judging from our cases and those in other reports, it is evident that ventral pressure against the dural sac by protruded disks can sometimes flatten and widen the spinal cord so as to mimic the myelographic appearance of an intramedullary tumor. Indeed, the gross appearance of the spinal cord at surgical exploration can simulate that seen with an intramedullary neoplasm.[3]

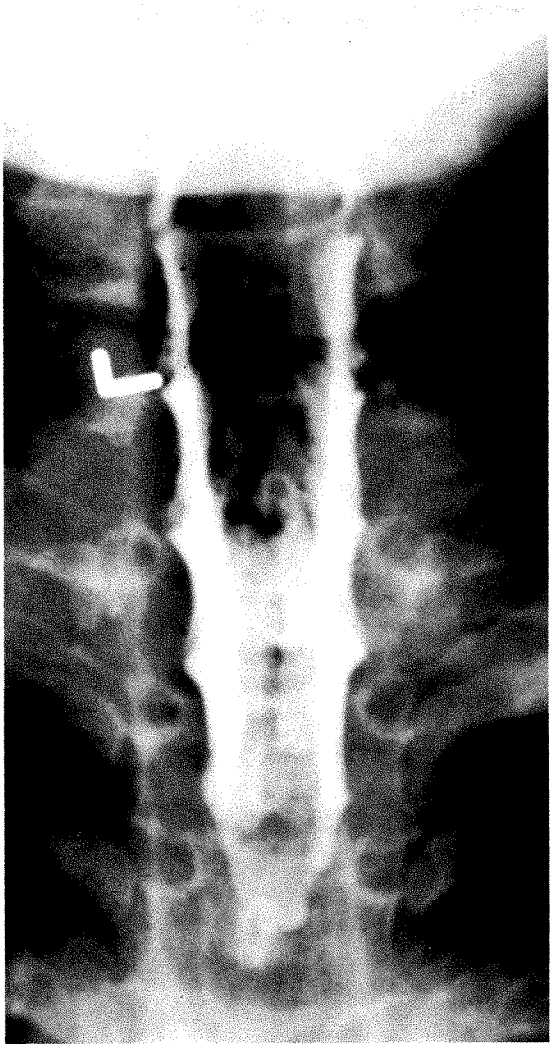


Fig. 4. Case II. Frontal myelogram shows the cervical spinal cord to be widened above the level of the seventh cervical vertebra. On this roentgenogram the cephalad extent of the cord widening is obscured by the occiput.

Wood[8] has previously noted that a midline cervical disk protrusion can cause a myelographic appearance which is indistinguishable from that of an intramedullary neoplasm in the frontal projection. Shapiro,[6] in his textbook, illustrates such a case and states that "the lateral film with the horizontal beam should help to establish the correct diagnosis." Schneider[5] reported the case of a middle-aged female, clinically

suspected of having a cervical cord tumor, in which cervical myelography revealed abrupt widening of the spinal cord from the fourth to the seventh cervical segment. Surgical exploration of the cervical spinal canal was performed, and extruded disk material was removed bilaterally from the space between the sixth and seventh cervical vertebrae; no cord neoplasm was present. A similar case, demonstrating on myelography an apparent cord tumor extending the length of three vertebral bodies, was reported by Yuhl and associates.[10] At laminectomy a flattened, widened cord was seen, secondary to multiple midline disk protrusions. After section of the dentate ligaments, the cervical cord regained a normal appearance.

Two myelographic features are useful in distinguishing intramedullary tumors from the cord widening produced by central disk protrusions. Both Wood[9] and Shapiro[6] have pointed out that short segments of cord widening, as in Case 1, are unusual with intramedullary tumors and are more suggestive of disk disease. In some cases of disk disease, however, the cervical cord is widened over a longer distance. The second and most valid myelographic differentiation between the two conditions is the appearance on the roentgenograms made in lateral position. With intramedullary tumors, the anteroposterior width of the cord is increased, sometimes filling the spinal canal. With cervical disk disease the anteroposterior width of the visible cord appears grossly normal. A ventral impression against the contrast column, opposite a disk space, can be seen at the level corresponding to the cord "enlargement" in the frontal projection.

SUMMARY

Clinical differentiation between cervical disk disease and spinal cord tumors may be difficult. In some cases of central disk protrusions, the myelographic appearance also can simulate a cord tumor if only frontal projections are employed. Two such cases are reported. The appearance on roentgenograms taken in lateral position made a correct preoperative diagnosis possible in both patients.

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THE EFFECT OF ANATOMIC VARIATIONS IN THE LUMBAR VERTEBRAE AND SPINAL CANAL ON CAUDA EQUINA AND NERVE ROOT SYNDROMES*

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THE size and configuration of the spinal canal and the intervertebral foramina constitute important factors in the production of symptoms referable to the cauda equina and nerve roots of the lumbar spine. The space available for the nerve trunks may determine whether or not an intrusion such as a disk herniation or a spondylotic ridge will or will not result in sufficient anatomic change to compromise the roots of the cauda equina as they pass from the spinal canal through the intervertebral foramina.

Verbiest[17,18] first recorded clinical observations regarding a radicular syndrome resulting from developmental narrowing of the lumbar vertebral canal. He regarded the relative stenosis as a manifestation of a general disturbance in skeletal growth rather than as an endogenously determined developmental problem. Symptoms resembling intermittent claudication with low back pain and sciatic radiation were the primary complaints. Neurologic changes were not prominent and were disproportionate as compared with the severe disability and pain. Acute symptoms were precipitated by small disk herniations and spondylotic spurs. Myelography disclosed partial or complete block in all of Verbiest's 13 patients. A rather characteristic defect was noted on the lateral myelograms, the column of contrast material ending in a sharp point at the level of the interspace blocked by this disorder. Schlesinger and Taveras[15] found that the size of the herniated disk in such individuals was secondary to the size of the spinal canal as the main cause of root compression. Postoperative traumatic paraplegia may occur in such patients because of a lack of appreciation of the structural peculiarities of narrow canals at the time of surgery.

The importance of taking into consideration the configuration of the spinal canal is emphasized by the usefulness of determining the sagittal diameter of the cervical spinal canal in the evaluation of disk disease in that region. A reduction in the normal sagittal diameter below 10 mm. to 12 mm. increases the possibility of cervical cord compression in the presence of spondylotic ridging or disk herniation.[1,13,19]

Normally, the lumbar spinal canal has a fairly consistent structural pattern. The vertebral foramen of the first and second lumbar vertebrae retain the somewhat rounded characteristics of the lower thoracic spinal canal. The vertical dimension is equal to or somewhat more than the interpedicular distance. The floor is slightly concave and rounded, its cephalic and caudal rims being slightly elevated. The lowermost three lumbar vertebrae have interpedicular dimensions exceeding the height of the canal.

MEASUREMENTS ON DRIED ANATOMIC PREPARATIONS

In measuring the widest dorsoventral diameter of the lumbar spinal canal in 50 dried vertebral columns, a slight but definite difference was noted in the elevation of the cephalad and caudad aspects of the neural arches. The latter is tilted upwards and is approximately 5 mm. higher than the cephalad margin. This permits

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overlapping or shingling of the respective apposing laminae so conspicuous in the cervical and thoracic vertebrae. The lateral portions of the vertebral foramina are important because the nerve trunks traversing the intervertebral foramina pass in close relationship to these recesses.[11] The lateral walls are formed by the convex medial surfaces of the pedicles. The lateral abutment of the laminae and the inferior margins of the superior and inferior articular facets cover the caudad and cephalad margins of the recesses and extend laterally to roof over the intervertebral foramina. A smooth groove is thereby formed for the passage of the nerve trunks. The roof of the intervertebral foramen is thus formed by the inferior aspect of the posterior articular facet and the immediately adjacent tip of the anterior articular facet. The lumbar pedicles are set in the upper half of the vertebral bodies. Inasmuch as the nerve trunks pass through the foramina closer to the cephalad portion, any constriction of the lateral recesses may be important in the genesis of nerve root compression.

Measurements were made of the lumbar spinal canal from the first lumbar to the lumbosacral levels. The points taken for the determination of the widest sagittal diameter were identified in relation to landmarks ascertained from the dried specimens, from plain spinal roentgenograms and erect full column myelograms. A slight discrepancy was found in measuring the widest diameter of the spinal canal in anatomic preparations because of the upward slope of the neural arches. The position of the dural sac in the canal is such that it is, for practical purposes, in contact with the floor in the upper three lumbar segments. At the fourth and fifth lumbar levels, a slight elevation of the dural sac from the floor of the canal is seen fairly often, more pronounced at the lumbosacral interspace, especially when the caudal sac is narrow and terminates in a pointed fashion at or slightly below this interspace. It should be recognized that variations in the lateral recesses cannot be identified directly from roentgenograms because no known projection brings these important compartments into view.

Measurements of the widest sagittal diameter of the lumbar vertebral foramina were made from 50 anatomic preparations at the American Museum of Natural History and those available to us in our own collection (Fig. 1, *A–E*). These measurements were correlated with the configuration of the lateral recesses of the respective vertebrae. It was found that the measurements corresponded fairly accurately with those made on erect full column myelograms, taking into consideration the magnification factor produced by the anatomic position of the spine as seen on lateral roentgenograms. Apparently, the space occupied by the epidural fat and vascular structures is sufficiently compressed when full column myelography is performed to permit fairly close approximation of the dural sac to the inferior aspect of the anterior portion of the neural arches. This was also observed on several vertebral columns inspected roentgenologically after passing thin Penrose drains for the entire length of the spinal canal and distending these to capacity with hypaque (Fig. 2, *A–C*). On comparing the plain roentgenograms of the spine with the full column myelograms and the various anatomic preparations described, it was found that a reasonable consistent measurement could be obtained by utilizing the uppermost level of the intervetebral foramen as a dorsal point and the floor of the spinal canal at the caudad or cephalad aspect as a ventral point. The depth of the canal is wider by 2 to 4 mm. if measurements are made in the hollow of the vertebral body's dorsal aspect.

It was thus ascertained that the normal values of the depth of the spinal canal varied between 1.5 and 2.3 cm. Measurements less than 1.5 cm. were considered below average, but little clinical significance was attached to measurements above 1.3 cm. As a rule these determinations can be made readily on accurate lateral roent-

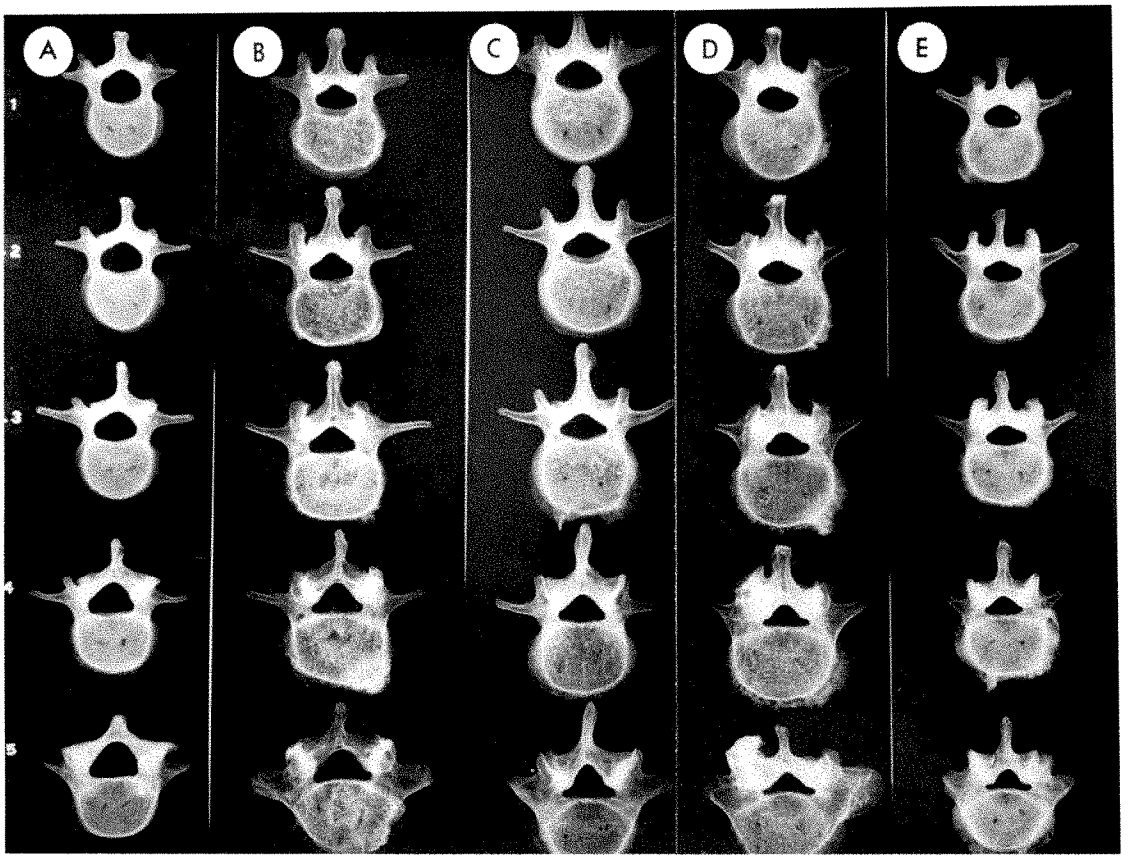
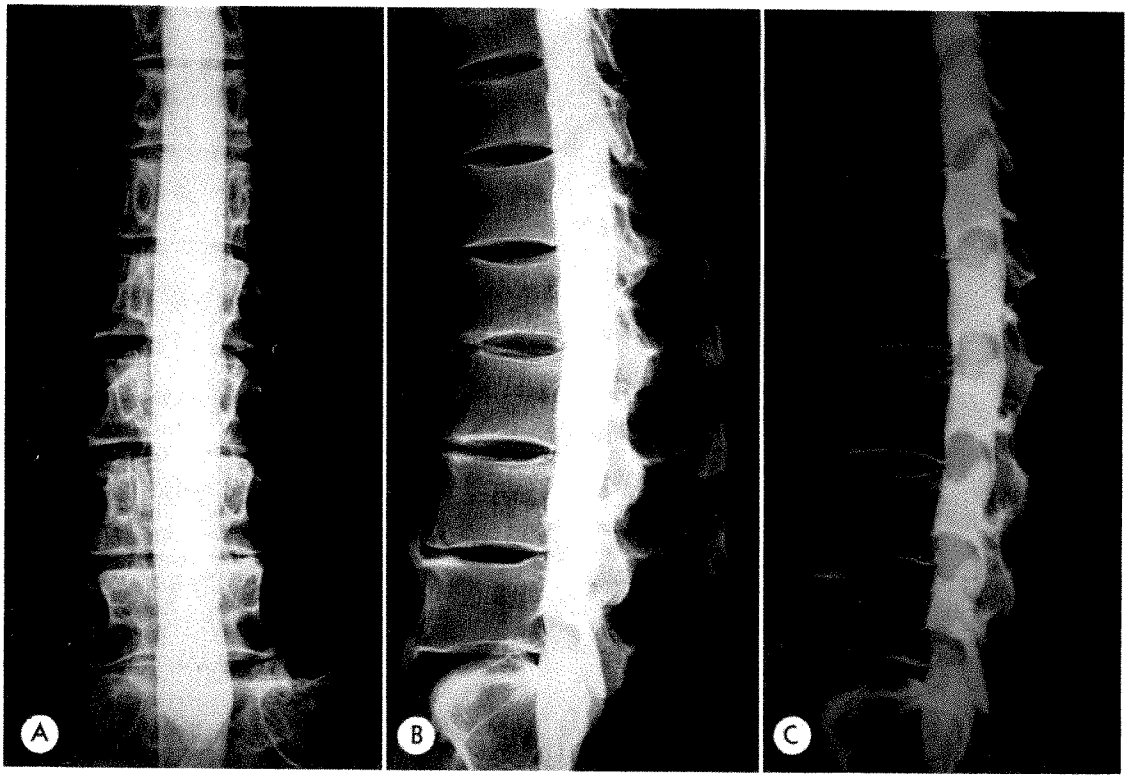


Fig. 1. Roentgenograms of five selected dried specimens of the lumbar vertebrae demonstrating variations in the vertebral foramina. The vertebral spinal foramina of the first and second lumbar vertebrae are more or less ovoid in configuration. In the lowermost three vertebrae a wide variability is observed in the vertebral foramina. Specimen *A* presents a wide fourth and fifth lumbar vertebral foramen. In *B*, *C*, *D* and *E*, the foramina are progressively narrowed in the sagittal diameter and the lateral recesses are attenuated. This process is most marked in specimens *D* and *E*, both of which have sagittal narrowing.

genograms of adequate detail. The measurements were made more accurately and with greater ease in the upper three lumbar levels, where they tended to be from 1 to 3 mm. more than at the fourth and fifth lumbar interspaces. The fourth lumbar interspace sometimes presented some difficulty, especially if the lateral projection was not accurately portrayed. This was even more noticeable at the lumbosacral interspace, especially if the intervetebral disk was narrowed or if scoliosis or some congenital malformation interfered with the accurate projection of the lumbosacral intervertebral foramen. Arthritic changes in the apophyseal joints also caused difficulty in identifying the superior rim of the intervertebral foramina when the articular margins were so blurred that they could not be identified clearly.

On correlating our observations of the configuration of the spinal canal, it was apparent that those vertebrae with narrowed sagittal diameters also had a definite tendency to significant constriction of the lateral recesses at the levels of the narrowing. It was also observed that the pedicles in these specimens were short and thick and that the neural arches were heavy. The articular facets were prominent and appeared vertically oriented, so much so that on the more seriously affected samples the medial margins approached the spinous processes, a circumstance also observed at


A
B
C

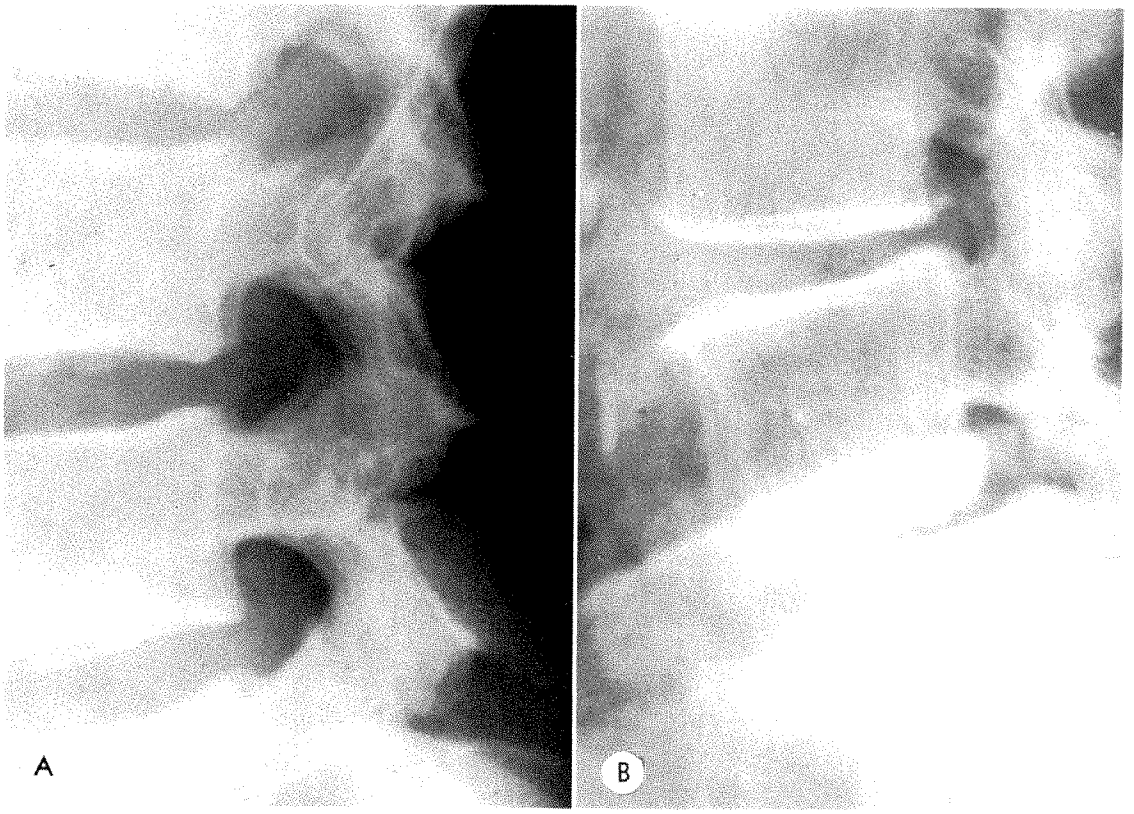

A
B

Fig. 2. (*A*) The lumbar canal of this dried specimen of the spine was threaded with a Penrose drain. Roentgenograms were made after the Penrose drain was distended to capacity with hypaque. The distended balloon does not quite reach the margins of the pedicles in the lumbar area. (*B* and *C*) The distended balloon has its superior surface in close approximation to the superior aspect of the respective intervertebral foramina.

operation. Narrowing of the entire lumbar canal was noted in 9 specimens, and in 12 others the narrowing was quite definite at the third and fourth interspaces only. In 3 the lowermost three lumbar vertebrae presented narrowed canals. These variations in vertebral architecture offered a reasonable explanation as to why seemingly small variants presented such a variable picture of nerve root compression. It also accounted for the necessity for adequate foraminal decompression as a requisite for satisfactory surgical results. The coexistence of spondylotic deformities also increases the possibility for intraforaminal or intraspinal disk disease to cause disproportionately prominent syndromes of root compression.

FACTORS CAUSING NARROWING OF INTERVERTEBRAL FORAMINA

In considering the available space in the lower lumbar spinal canal, account should be taken of the fact that sagittal narrowing and attenuation of the lateral recesses and intervertebral foramina are but part of the picture. Other factors include thickening of the neural arches,[9] spondylotic ridges and spurs[3–6] along the posterior vertebral margins and the articular facets, spondylolisthesis (Fig. 3, *A* and *B*), and thickening of the ligamenta flava.[12] Congenital variations in the neural arches, particularly at the lumbosacral interspace, also may diminish the space for the caudal sac and the emerging nerve roots.[2,10] The effect of variations in posture on the configuration of the spinal canal rests mainly on the changes produced in the intervertebral disks and ligamenta flava. Cough or strain also causes change in the available space because of engorgement of the prominent epidural venous plexuses. This can be seen often during myelography when the patient strains. The pantopaque column becomes narrowed and pushes cephalad for one or two interspaces.[2,14] In the presence of a narrowed lumbar canal, all of these factors may contribute to sufficient loss of available space to make even a minor disk protrusion or a small spondylotic spur, strategically situated, clinically important. Sim-

Fig. 3. (*A*) Lateral roentgenogram of a normal vertebral column in an 18 year old male patient demonstrating the normal configuration of the third and fourth lumbar intevertebral foramina. (*B*) Lateral roentgenogram of the lower lumbar spine in a 55 year old patient with spondylolisthesis of L_4 and L_5. The intervertebral foramina are significantly narrowed. The fourth intervertebral foramen is further compromised by the shift incident to the spondylolisthesis.

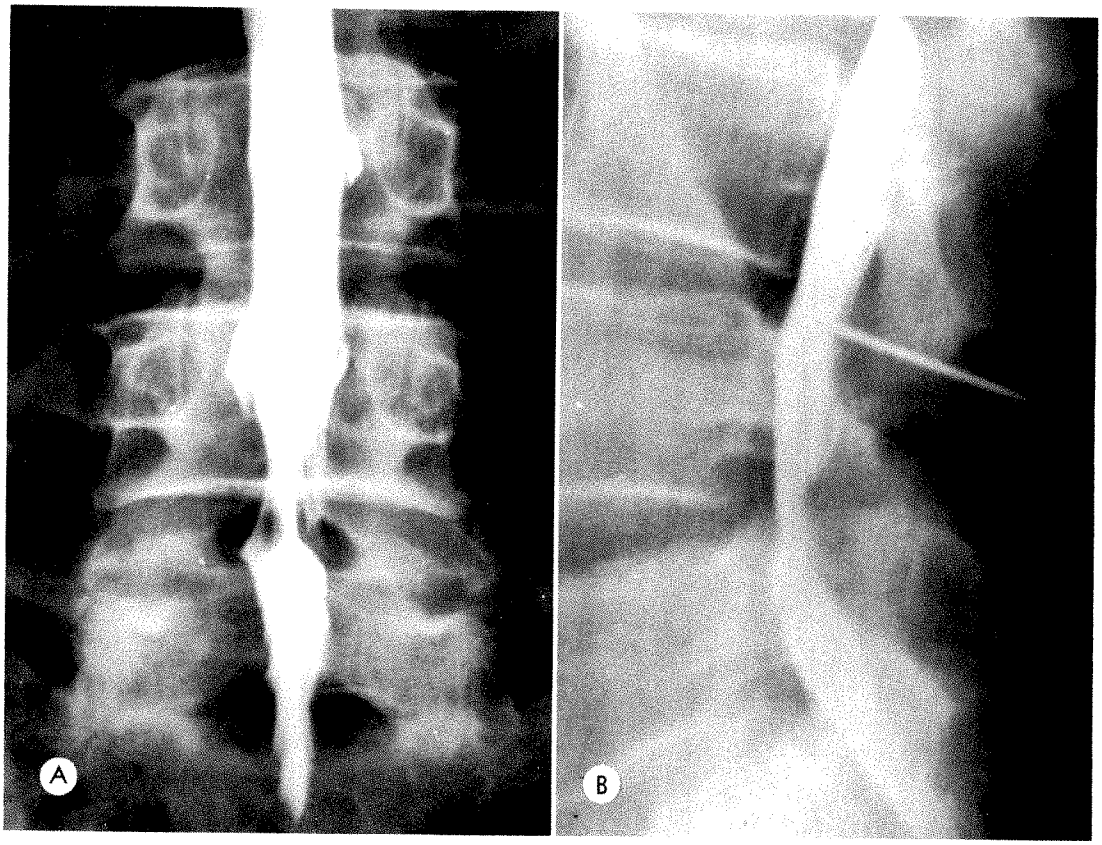


FIG. 4. (*A*) Anteroposterior full column myelogram of a 57 year old man with severe low back pain with left sciatic radiation. Pain was aggravated by strain and postural changes. Note the waist-like configuration in the pantopaque column at the fourth lumbar interspace. (*B*) On the lateral myelogram there is a relatively sharply localized defect at the fourth lumbar interspace. The pantopaque column is compressed in its ventral aspect by elevation of the posterior spinal ligament. Its dorsal aspect is impinged upon by thickening of the neural arch. The fourth lumbar intervertebral foramen is narrowed. At operation a herniated disk was found at the fourth lumbar interspace. The elevation of the thecal sac was due to extrusion of the disk tissue, of which about 1 cc. was removed. The canal was markedly narrowed, the dorsal aspect being compromised by the heavily thickened laminae and hypertrophied ligamenta flava. The lateral recesses were markedly narrowed. The defect apparently was due to the combination of local malformation of the spinal canal and relatively minor disk herniation. The patient made a good recovery following laminectomy and wide foraminotomy.

ilarly, bulging of a degenerated disk without actual extrusion of cartilage may result in root compression.

CLINICAL MATERIAL

The present report derives from a clinical study of 29 patients with symptoms of nerve root compression causally related to narrowing of the lumbar spinal canal.[7] The majority of patients were in the fifth and sixth decades of life. Radicular pain was the most conspicuous symptom, often so intense as to suggest hysteria. The onset of symptoms was related to stress and injury in 13. No characteristic body habitus was observed. The most seriously disabled patients complained of bilateral lower extremity pain. Sensory alterations and weakness were noted as pain increased. Sphincter dysfunction occurred in only 1 patient. Reflex changes were present in 22, including absence of the ankle jerk in most cases, a depression of the patellar reflex to a lesser degree in others. Both the fifth lumbar and

first sacral dermatomes were affected by the sensory alterations, 3 patients having involvement of the saddle area. Weakness was most evident in the distal musculature, extending to foot drop in those most seriously affected.

The difficulties encountered in the performance of lumbar puncture often aroused suspicion of narrowing of the canal. The depth of the spinal canal varied from 0.8 to 1.5 cm. in these patients. A defect in the dorsal aspect of the pantopaque column was related to inward protrusion of the ligamenta flava and the thickened laminae. Varying bilateral waist-like defects were noted in the frontal projection, in some instances producing a partial block. In a few instances the block was relatively complete, and in these a characteristic pointed defect was seen at the level of the involved space on the lateral myelograms as described by Verbiest. These defects were most frequent at the third and fourth lumbar interspaces, often resembling those found in the presence of advanced spondylosis of the vertebral bodies (Fig. 4, *A* and *B;* and 5, *A–C*). In a few instances arthritic changes were present in the apophyseal joints as well.[4,5] Initial interpretations of some of the earlier examinations included arachnoiditis, cauda equina tumor and extruded intervertebral disk. As sufficient data were accumulated, we recognized the significance of associating the shape of the spinal canal with the myelographic defects.

Surgical decompression is essential and laminectomy over multiple levels often is indicated. Foraminal decompression and excision of intruding osteophytes and fragments of disk material complete the procedure. Certain individuals may show unusually prominent vertically oriented pos-

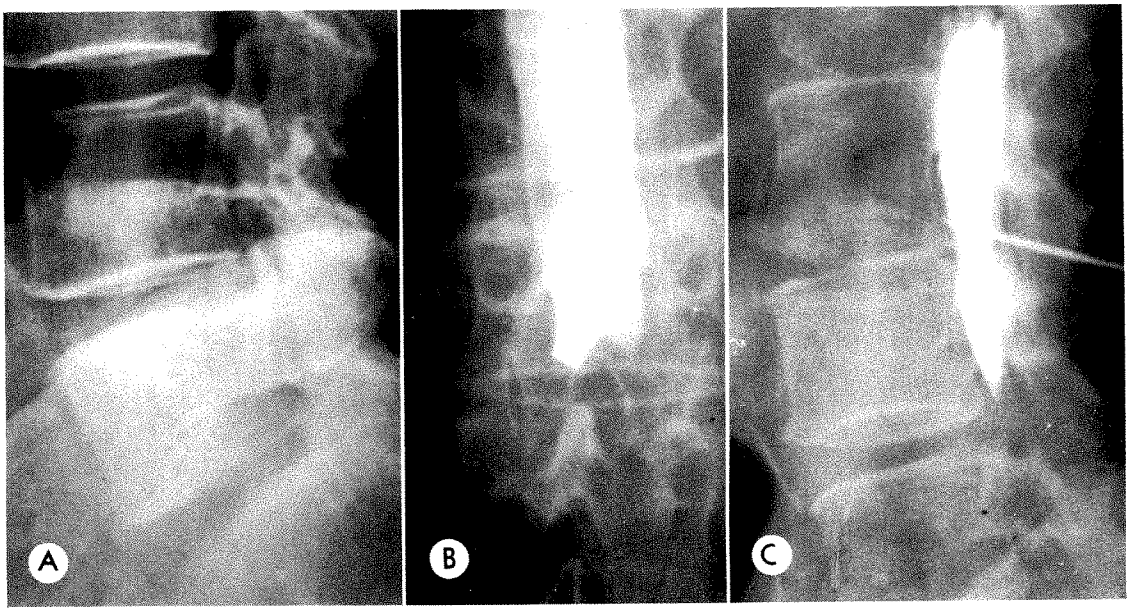


Fig. 5. (*A*) Lateral roentgenogram of lower lumbar spine of a 72 year old woman who had severe sciatic pain for 6 months, aggravated by exertion. The intervertebral foramina are elongated and narrowed. The laminae are diffusely thickened. (*B*) Same patient. Anteroposterior myelogram shows a complete block at the level of the inferior margin of the fourth lumbar vertebra. (*C*) On the lateral myelogram the pantopaque column assumes a pointed appearance caudally. At operation, the spinal canal was markedly narrowed and the lateral recesses were sharply attenuated. Slight bulging of the intervertebral disk was sufficient to cause a complete block. The patient was relieved following laminectomy, foraminotomy and excision of the degenerated disk matter. The configuration of the pantopaque column, particularly the tapered caudal end of the column, corresponds with the appearance described by Verbiest.[17,18] (Reproduced by permission from *J. Neurology, Neurosurg. & Psychiat.*, 1962, *25*, 163.)

terior articulations. The laminar thickening and the imbrication of these structures may render the operation rather difficult, but should serve to alert the surgeon to the necessity for adequately decompressing the narrowed areas, especially the lateral recesses and intervertebral foramina involved.

COMMENT

Sagittal narrowing of the spinal canal occurs as a congenital malformation in achondroplasia.[8,16] In these patients the vertebral bodies are flattened, but otherwise are quite large. The pedicles, however, are short and the intervertebral foramina are narrow and elongated. The laminae are usually heavy and their posterior aspects have a definite tendency to converge. It is in these patients that relatively small defects may produce inordinate symptoms, and relief may be obtained only by wide laminectomy.[8]

The question as to whether the diminution in the sagittal diameter of the spinal canal in otherwise apparently normal individuals may have a congenital basis logically comes up for consideration. It would appear reasonable to assume that variations in the configurations of spinal canals occur, just as variations in other aspects of vertebral architecture are observed. We have seen young patients with herniated disks and symptoms out of proportion to the amount of herniation present. The spinal canals in these patients were narrowed and the lateral recesses, as observed at operation, were attenuated so that the nerve roots were severely impinged upon even in the absence of major portrusions. On the other hand, it has been noted that large myelographic defects may occur in patients who have no symptoms referable to that level. It is postulated that sufficient available space exists in these patients so that there is no nerve root compression. Impingement on the dural sac alone does not necessarily produce symptoms. The defect in the myelograms indicates the inward protrusion of a spur or disk herniation. If such a defect is not associated with compression of a nerve root or a significant degree of block in the spinal canal, symptoms may be absent. Indeed, we have seen occasional instances of patients with large defects in the lumbar area approaching complete block without symptoms referable to the spondylotic spurring obviously present. These patients had myelographies performed for symptoms referable to the thoracic or cervical regions, and it was important to carry the pantopaque to the suspected clinical levels rather than trust the dramatic but otherwise insignificant defects seen in the lumbar canal.

Spondylosis deformans may be an important contributing factor to the production of symptoms referable to the cauda equina and its roots because of pressure. These ridges often are present at the dorsolateral portions of the vertebrae in the floor of the spinal canal where they may contribute to diminution of the available space. Spondylotic changes along the articular facets and the inferior aspects of the laminae also may be important. A relatively small protrusion so placed that it impinges into the intervertebral foramen may be far more significant than a large spur not in contact with a nerve root. It is important to remember that a spondylotic spur projecting into the spinal canal in a patient with a narrowed sagittal diameter in the lumbar vertebral foramina is quite likely to cause symptoms referable to this disorder. Unless these intrusions are removed at operation and the lateral recesses decompressed, there may be unsatisfactory results.

Plain roentgenograms provide a clue as to the sagittal diameter by evaluation of the height of the intervertebral foramina. In those patients with narrowed canals, as indicated by foraminal heights less than 13 mm., it has been found that spinal puncture sometimes is difficult at the usual third and fourth interspaces even if the tap is performed skillfully with the needle tip properly directed to the midline. This occurs more often with pronounced narrow-

ing such as occurs in patients with achondroplasia. In others, it may be noted that subarachnoid instillation of 6 cc. of pantopaque fills the canal so that on erect examination the column reaches the third lumbar level. It has been our experience that while the introduction of pantopaque into these patients may be fairly uneventful, its removal often is difficult. In these patients we have found it expedient to permit the patient to express the contrast material by straining rather than by aspiration. If this does not work and if the lumbosacral portion of the caudal sac is larger, a fifth lumbar tap may be tried, hoping that a direct midline puncture would permit easier removal of the contrast material.

SUMMARY

Measurement of the height of the lumbar intervertebral foramina permits a fairly accurate evaluation of the sagittal diameter of the spinal canal. Measurements vary between 1.5 and 2.3 cm. normally. Those below 1.3 cm. are considered abnormal. In these patients small protrusions, either from disk herniations or spondylotic spurs, or both, may produce disproportionately severe symptoms because of diminution of the available space for the nerve roots in the lateral recesses of the spinal foramina involved.

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DIAGNOSTIC IMPLICATIONS OF RIB NOTCHING

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NOTCHING of the inferior borders of the ribs was first described as a roentgenologic sign of coarctation of the aorta by Roesler[19] in 1928. For a number of years thereafter, this sign was believed to be pathognomonic of coarctation. In more recent years, both unilateral and bilateral rib notching have been described secondary to various other conditions. The detection of rib notching on roentgenograms of the thorax, therefore, may have diagnostic implications of considerable significance. The conditions in which rib notching has been described are listed and classified in Table 1.

Although rib notching has been described in each of the conditions listed (Table 1), not much difficulty should be encountered in determining the cause of the notching when this roentgenologic finding is encountered. The characteristics of the rib notching in each condition and the associated findings that may be present permit the radiologist to make an accurate diagnosis in most instances. When these roentgenologic findings are considered along with the clinical history and physical findings, the diagnosis becomes readily apparent in most cases.

The purpose of this paper is to describe the various causes of unilateral or bilateral rib notching and to discuss some of the important diagnostic implications of this roentgenologic finding.

TABLE I

CAUSES AND CLASSIFICATION OF RIB NOTCHING

Coarctation of the aorta
Interruption or obstruction of the subclavian or innominate arteries
Blalock-Taussig operation
Atheromatous or thrombotic occlusion
Pulseless disease (Takayasu's disease)
Conditions with obstruction to pulmonary blood flow
Cardiac malformations with pulmonary stenosis or atresia
Agenesis of a main branch of the pulmonary artery
Occlusion of a lobar branch of the pulmonary artery
Arteriovenous fistulas of the lung or chest wall
Pulmonary arteriovenous fistula
Systemic-to-pulmonary arterio-arterial or arteriovenous fistula
Systemic-to-systemic arteriovenous fistula of the chest wall
Superior vena caval, innominate, or subclavian vein obstruction
Intercostal nerve tumors
Neurofibromatosis
Other neurogenic tumors
Other reported causes
Arteriosclerosis, hypertension, and conditions causing an increased systemic pulse pressure
"Idiopathic" rib notching
"Pseudo" rib notching in tuberous sclerosis

COARCTATION OF THE AORTA

Coarctation of the aorta is by far the most common cause of rib notching. The rib notching in coarctation is the result of dilatation, tortuosity, and increased pulsations of the intercostal arteries because of increased collateral flow through these vessels (Fig 1 and 2). The erosion occurs not at the inferior margin of the rib, which is often invisible on the roentgenogram, but rather in the costal groove on the lower anterior surface of the rib (Fig. 2). The rib notching in coarctation is bilateral in most cases (Fig. 3). Characteristically, the fourth to eighth ribs are involved; infrequently, the third and ninth ribs show evidence of notching. In rare cases in which the coarctation is located in the lower thoracic or abdominal aorta, notching may occur in the lower ribs but may be absent in the midthoracic ribs (Fig. 4, *a* and *b*). In coarctation, the collateral circulation often is not well developed, and rib notching does not occur. Holmes[11] reported rib notching in

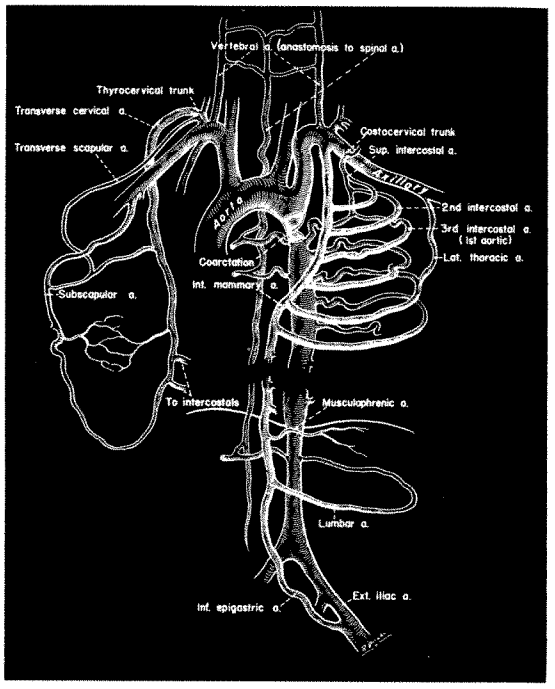


Fig. 1. Main channels of collateral circulation in coarctation of aorta. Edwards, J. E., Dry, T. J., Parker, R. L., Burchell, H. B., Wood, E. H., and Bulbulian, A. H. An Atlas of Congenital Anomalies of the Heart and Great Vessels. Charles C Thomas, Publisher, Springfield, Illinois, 1954.

192 of 300 patients with coarctation of the aorta. Rib notching in coarctation is uncommon before adult life. The youngest patient with coarctation in whom we have observed rib notching was 4 years of age.

Rib notching in coaractation may have important implications in regard to the location of the coarcted segment in relation to the origin of the subclavian arteries as well as in patients with coarctation of the lower thoracic and abdominal aorta. Unilateral rib notching on the right in coarctation may be ascribed to coarctation proximal to the origin of the left subclavian artery or associated with stenosis or atresia of the left subclavian artery (Fig. 5). In such cases, the left subclavian artery is a low pressure artery, and collateral circulation develops only on the right. In patients with coarctation of the aorta proximal to the origin of an aberrant right subclavian artery, the aberrant right subclavian artery, arising as the most distal branch of the aortic arch, is a low pressure artery, and rib notching occurs on the left side only (Fig. 6).



Fig. 2. Rib and dilated and tortuous intercostal artery removed at necropsy in a case of coarctation of the aorta showing characteristic rib erosion. Edwards, J. E., Dry, T. J., Parker, R. L., Burchell, H. B., Wood, E. H., and Bulbulian, A. H. An Atlas of Congenital Anomalies of the Heart and Great Vessels. Charles C Thomas, Publisher, Springfield, Illinois, 1954.

We have observed one patient who had a right-sided aortic arch and an aberrant left subclavian artery arising as the fourth branch of the arch in whom coarctation was present proximal to the origin of the aberrant left subclavian artery. In this patient, as might be expected, unilateral rib

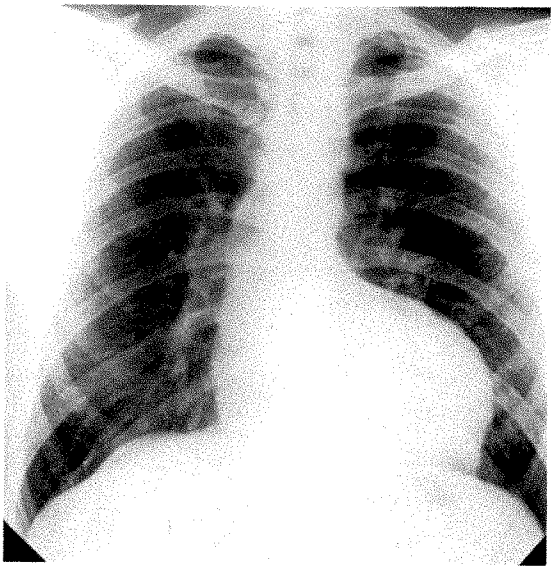


Fig. 3. Bilateral rib notching in a 28 year old man with coarctation of aorta. Left ventricle is enlarged and the characteristic figure-of-three sign is present, indicating site of coarctation. Ascending aorta is dilated.

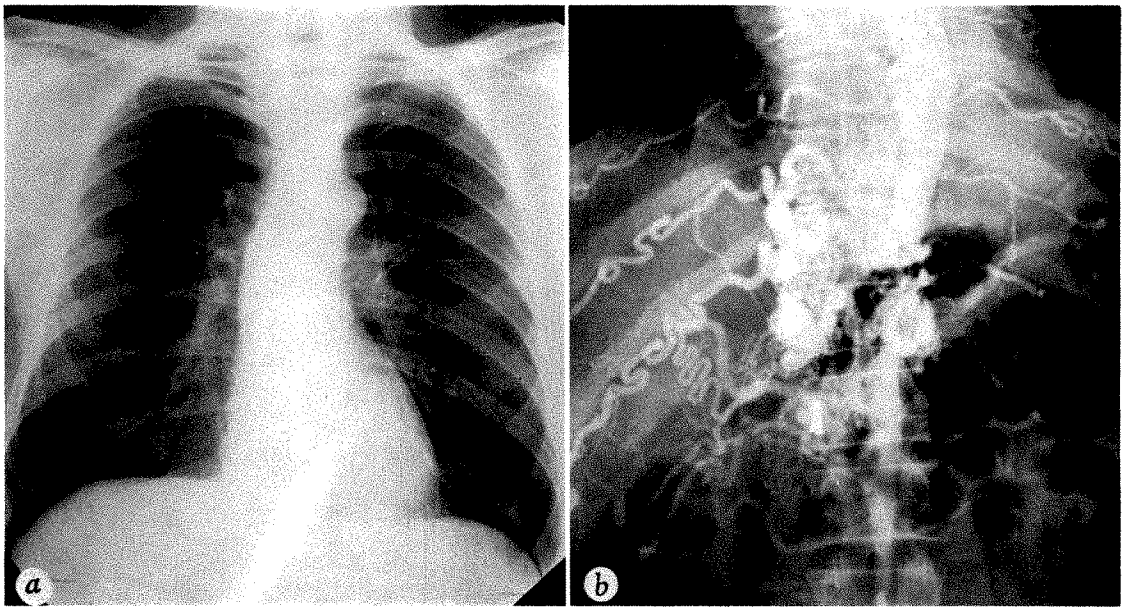


FIG. 4. Fifty-five year old man with clinical signs of coarctation of the aorta. (*a*) Notching of lower ribs without notching of the mid-thoracic ribs that is usually seen in coarctation of aorta. (*b*). Thoracic aortogram showing severe coarctation of upper abdominal aorta. Lower intercostal arteries are dilated and tortuous.

notching was present on the right (Fig. 7, *a* and *b*).

In most cases, the coarctation lies distal to the ductus arteriosus and the ductus obliterates normally. When coarctation occurs proximal to a patent ductus arteriosus, adequate circulation to the lower body may be maintained by means of a right-to-left shunt through the ductus. In such cases, rib

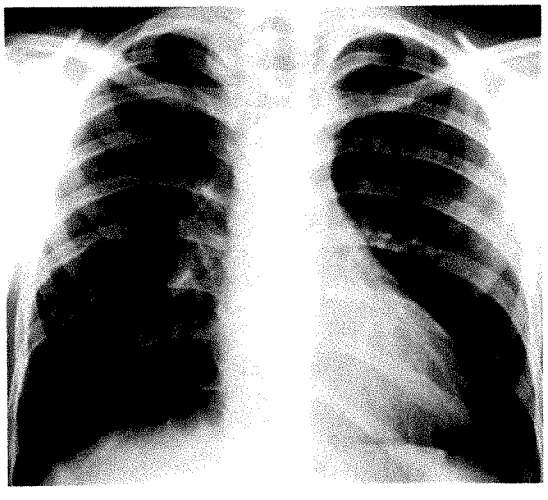


FIG. 5. Coarctation of aorta in a 9 year old boy. Left subclavian artery arose from aorta at site of coarctation and orifice of this vessel was stenosed. Rib notching is present only on the right.

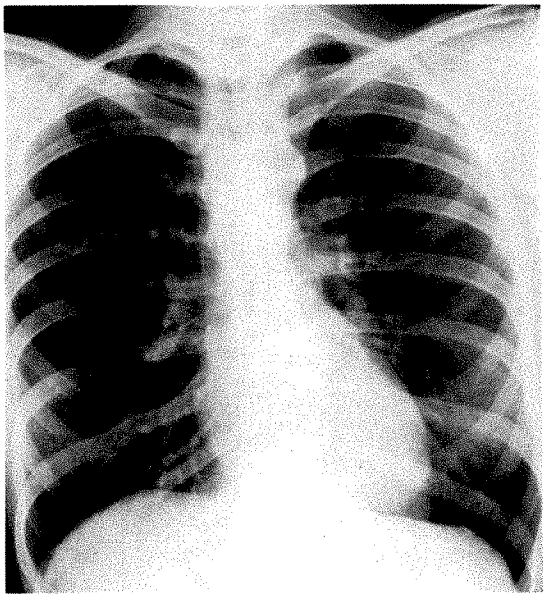


FIG. 6. Coarctation of aorta in a 27 year old woman. Aberrant right subclavian artery originated distal to coarcted segment. Only left ribs are notched.

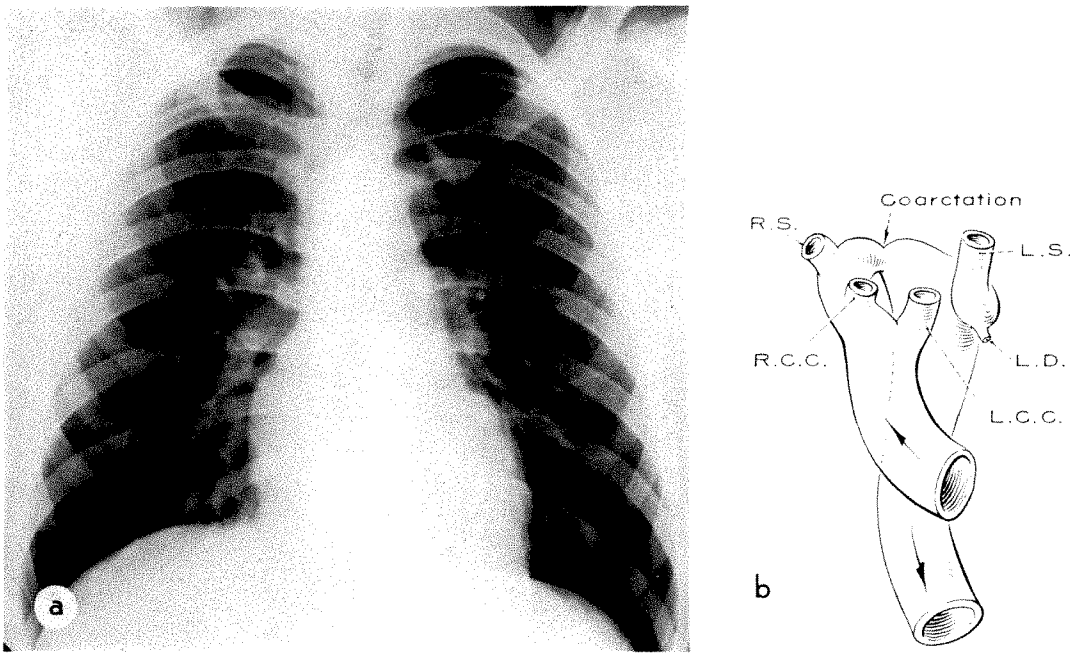


Fig. 7. (*a*) Minimal notching of mid-thoracic ribs on right in a 16 year old boy with right aortic arch and coarctation of the aorta proximal to origin of aberrant left subclavian artery. (*b*) Diagram of anomaly. (R.C.C. = right common carotid artery, R.S. = right subclavian artery, L.S. = aberrant left subclavian artery, L.D. = left ductus arteriosus, L.C.C. = left common carotid artery.) Stewart, J. R., Kincaid, O. W., and Edwards, J. E. An Atlas of Vascular Rings and Related Malformations of the Aortic Arch System. Charles C Thomas, Publisher, Springfield, Illinois, 1964.

notching usually does not occur since a well-developed collateral circulation bypasses the coarctation.

When associated signs such as left ventricular enlargement and the characteristic figure-of-three sign indicating the site of the coarctation are present (Fig. 3) and when other lesions are not evident in the lungs or chest wall, the cause of the rib notching will be readily apparent.

Interruption or Obstruction of the Subclavian or Innominate Arteries

Blalock-Taussig Operation. Unilateral rib notching commonly occurs secondary to interruption of a subclavian artery for the Blalock-Taussig subclavian-pulmonary artery anastomosis. The intercostal arteries take part in the collateral circulation to the arm after division of the subclavian artery. Blood flow in such cases is in the opposite direction to that of the collateral circulation in coarctation of the aorta. In these cases, blood flow is from the descending thoracic aorta, through the intercostal arteries and the lateral thoracic plexus to the axillary artery.

Rib notching after the Blalock-Taussig operation was first described by Kent[13] in 1953. Since that time, numerous reports have dealt with this subject. It has been proposed, and is commonly believed, that rib notching after the Blalock-Taussig operation is indicative of closure and nonfunction of the subclavian-pulmonary artery anastomosis. We have seen a number of patients, however, who exhibited unilateral rib notching after the Blalock-Taussig operation in whom the anastomosis was functioning well. The development of rib notching in such cases is adequately explained by the development of collateral vessels secondary to interruption of the subclavian and vertebral arteries on the involved side and is in no way related to the function of the anastomosis.

Atheromatous or Thrombotic Occlusion. Although it has been frequently stated that it is reasonable for rib notching to occur in cases of long-standing atheromatous or thrombotic occlusion of the subclavian artery, we have encountered no such cases and, to our knowledge, none has been reported in the literature. We think that there is a logical explanation for the absence of rib notching in such cases. It probably can be explained on the basis that such occlusion almost invariably occurs in the proximal portion of the subclavian artery, proximal to the origin of the vertebral artery. In this event, good blood flow to the arm with normal or near normal pressure in the brachial artery may be found since the blood supply to the arm is maintained through the vertebral arterial system. In such cases, blood from the opposite subclavian artery passes through the vertebral artery that arises from this vessel and then in reverse direction through the vertebral artery on the involved side to supply the subclavian artery distal to the site of obstruction (so-called subclavian steal syndrome). This vertebral collateral pathway does not function after the Blalock-Taussig operation because in this operation the vertebral artery is ligated and interrupted to permit greater mobility of the subclavian artery.

Vaughan,[23] in 1959, reported a case of thrombotic occlusion of the innominate artery in which thoracic aortography showed large tortuous intercostal arteries on the involved side. Rib notching, however, was not demonstrated in this case.

Pulseless Disease (Takayasu's Disease). Weir and Kyle,[24] in 1956, reported a case of chronic obliterative arteritis of the subclavian and carotid arteries, so-called "pulseless disease" or "Takayasu's disease," in which bilateral rib notching was present, simulating that seen in coarctation of the aorta. Rib notching is evidently rare in such cases. It might be expected to occur more often when bilateral occlusion of the subclavian arteries is present, since in these cases the "subclavian steal" phenomenon does not exist to provide collateral circulation.

CONDITIONS WITH OBSTRUCTION TO PULMONARY BLOOD FLOW

Since the intercostal arteries may take part in collateral circulation to the lungs whenever there is obstruction to pulmonary blood flow, it is expected that rib notching would be seen occasionally in such conditions.

Cardiac Malformations With Pulmonary Stenosis or Atresia. The most common such condition is the tetralogy of Fallot. In many cases of severe tetralogy, collateral circulation to the lungs through the intercostal arteries, bronchial arteries, inferior phrenic arteries, and other vessels in the mediastinum is abundant and well developed. In spite of this well-developed collateral circulation, we have not observed rib notching in any case of tetralogy of Fallot except after a Blalock-Taussig operation.

Batchelder and Williams,[1] in 1948, reported a case of tetralogy of Fallot in which rib notching occurred on the left side. The rib erosions were found at necropsy to be due to dilated and tortuous intercostal arteries. Campbell,[5] in 1958, reported 2 cases of tetralogy of Fallot, 1 of pulmonary atresia and 1 of Ebstein's malformation, in which rib notching was present.

One patient with transposition of the great arteries and valvular and subpulmonary stenosis, which we have studied by means of selective angiocardiography, exhibited minimal but definite rib notching.

Agenesis of a Main Branch of the Pulmonary Artery. We have observed 1 patient with agenesis of a main branch of the pulmonary artery and rib notching on the involved side (Fig. 8). This case has been previously reported by Sherrick, Kincaid, and DuShane.[20] The rib notching in this case can be explained on the basis of increased collateral flow through the intercostal arteries on the side of the pulmonary artery agenesis. A similar case has been reported by Felson.[8]

Occlusion of a Lobar Branch of the Pul-

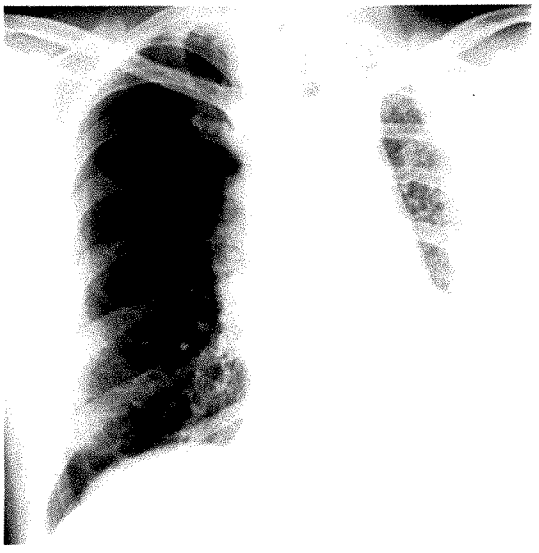


Fig. 8. Agenesis of left pulmonary artery and right-sided aortic arch with notching of left fifth and sixth ribs in a 59 year old man.

monary Artery. We have also observed a $3\frac{1}{2}$ year old boy with occlusion of the apical posterior branch of the right upper lobe pulmonary artery who exhibited unilateral rib notching (Fig. 9, *a–d*). Angiograms in this case revealed both the occlusion of the pulmonary artery branch and the enlarged tortuous right third and fourth intercostal

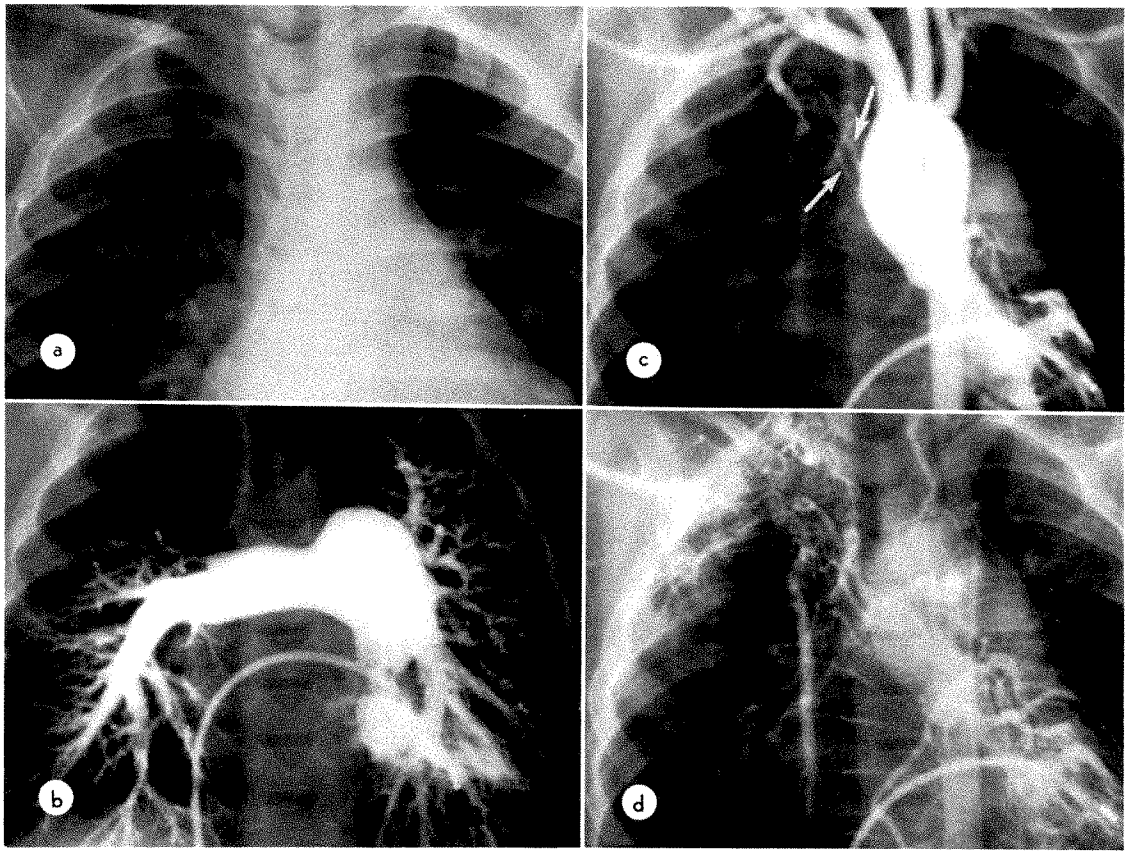


Fig. 9. Three and one-half year old boy with transposition of great arteries, referred following anastomosis of the azygos vein to the right apical posterior pulmonary artery and creation of atrial septal defect. (*a*) Roentgenogram made 3 years after operation. Notching of third and fourth ribs on right. (*b*, *c* and *d*) Angiocardiograms made with injection into left ventricle, which gives rise to the pulmonary artery. (*b*) Occlusion of apical posterior branch of the right upper lobe artery is shown. (*c*) Note early opacification of large right third and fourth intercostal arteries (arrows). (*d*) Exposure made 2 seconds after *c* showing large, tortuous intercostal arteries and other collateral vessels in vicinity of notching.

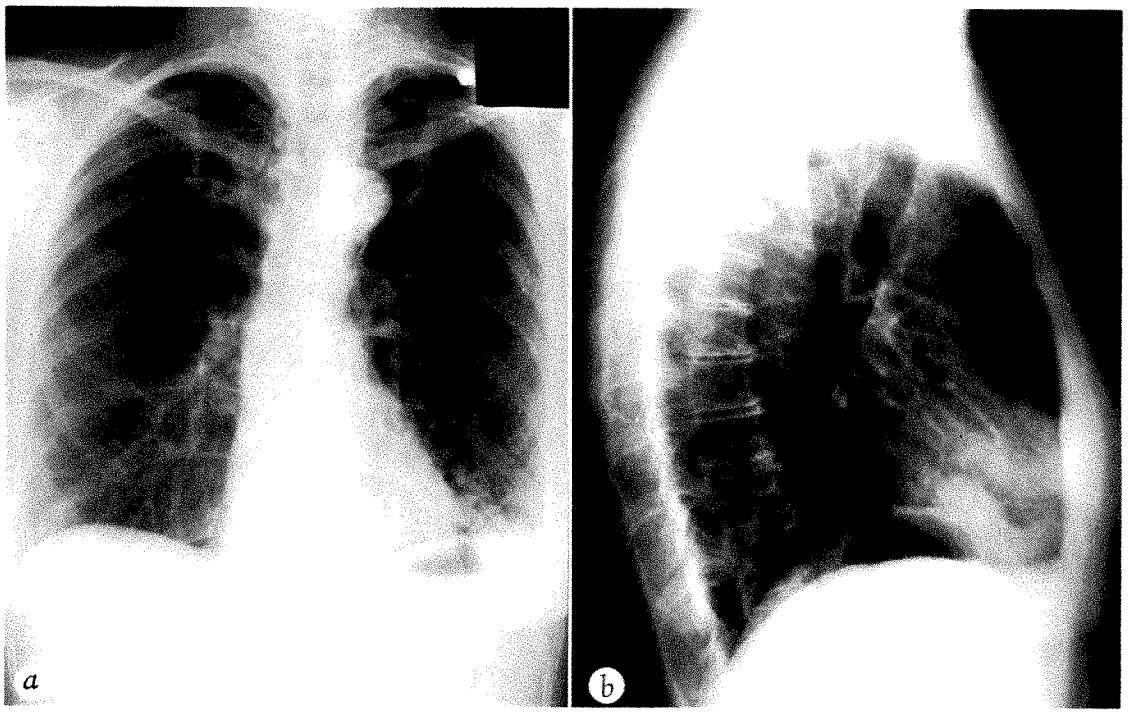


FIG. 10. Arteriovenous fistula in base of left thorax of a 54 year old woman.[9] The shunt was from the intercostal arteries to the pulmonary vascular system. There is notching of the seventh through the tenth ribs on left. (*a*) Posteroanterior view. (*b*) Lateral view.

arteries at the site of the rib erosion. This patient is the youngest in whom we have observed rib notching from any cause.

ARTERIOVENOUS FISTULAS OF THE LUNG OR CHEST WALL

Pulmonary Arteriovenous Fistula. Stork,[21] in 1955, described 2 cases and Prutzman and Flick,[18] in 1954, 1 case of pulmonary arteriovenous fistula in which unilateral rib notching was present. In 1960, Wilson[25] described another such case in which notching of multiple ribs on the involved side was noted. The rib notching in all 4 cases has been ascribed to the presence of dilated intercostal arteries carrying a systemic supply to the pulmonary arteriovenous fistula. It seems possible that in some cases the dilated intercostal arteries may contribute collateral circulation to that portion of the pulmonary vascular bed bypassed by the large flow through the pulmonary arteriovenous fistula.

Systemic-to-Pulmonary Arterio-arterial or Arteriovenous Fistula. Systemic-to-pulmonary arterio-arterial or arteriovenous fistulas occur as abnormal communications between high pressure systemic arteries and low pressure pulmonary arteries or veins. The systemic or afferent arteries may be bronchial arteries that enter the fistula from the mediastinum, or systemic arteries of the chest wall that traverse the pleural space over the periphery of the lung to supply the fistula. In the latter type, there may be increased vascularity of the chest wall and frequently marked enlargement of the intercostal arteries which contribute a blood supply to these fistulas. Rib erosion is not uncommon in such cases. Two cases of systemic-to-pulmonary arteriovenous fistula involving the vessels of the chest wall in which rib notching was present have been encountered at the Mayo Clinic. One of these cases has been reported by Good[9] (Fig. 10, *a* and *b*). Roentgenograms of the second case are shown in Figure 11, *a* and *b*.

Unilateral rib notching can be seen on the

roentgenogram of the thorax in the similar case reported by Brain and Kauntze.[3]

Systemic-to-Systemic Arteriovenous Fistulas of the Chest Wall. Arteriovenous fistulas that involve the intercostal vessels or other vessels of the chest wall also may produce rib notching. Unilateral rib notching due to an intercostal arteriovenous fistula was first reported by Hench and Horton[10] from the Mayo Clinic in 1933 (Fig. 12). Since that time, we have observed 1 additional case of unilateral rib notching that resulted from dilated intercostal arteries in a chest-wall arteriovenous fistula. This case was reported by Takaro and Clagett[22] in 1951 (Fig. 13). A case of unilateral rib notching secondary to an intercostal arteriovenous fistula also was described by Maier and Stout[15] in 1950.

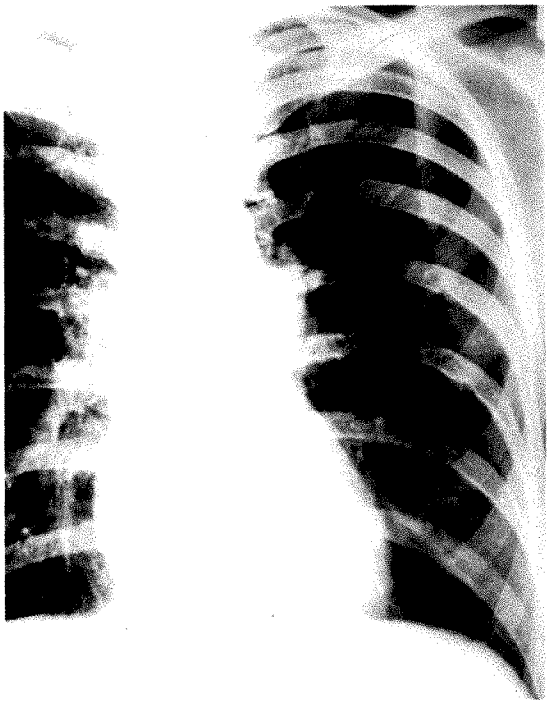
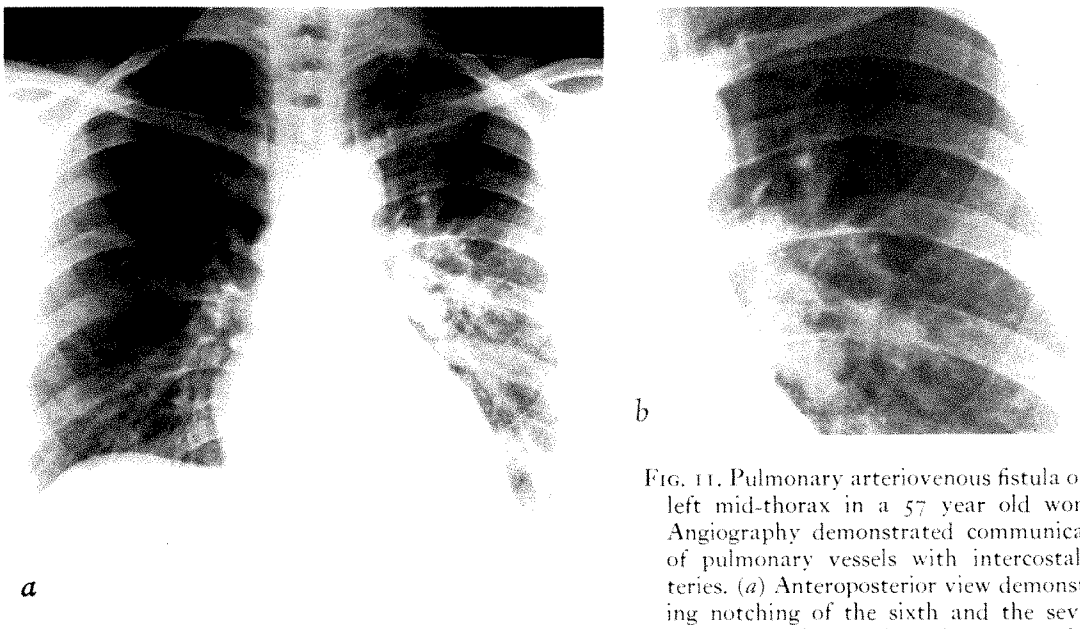


FIG. 12. Posteroanterior view showing notching of left seventh and eighth ribs. Patient was a 33 year old woman with arteriovenous fistula of intercostal vessels of left posterior thorax. A loud bruit could be heard in left seventh intercostal space. (Reproduced with permission from *M. Clin. North America.*[10])

OBSTRUCTION OF SUPERIOR VENA CAVA, INNOMINATE, OR SUBCLAVIAN VEIN

Cases have been described in which long-standing obstruction to the superior vena cava, innominate vein, or subclavian vein has resulted in rib notching. Presumably, bilateral rib notching may occur in cases of superior vena caval obstruction, while the



a *b*

FIG. 11. Pulmonary arteriovenous fistula of the left mid-thorax in a 57 year old woman. Angiography demonstrated communication of pulmonary vessels with intercostal arteries. (*a*) Anteroposterior view demonstrating notching of the sixth and the seventh ribs on left. (*b*) Localized view of left side of thorax demonstrating the notching in greater detail.

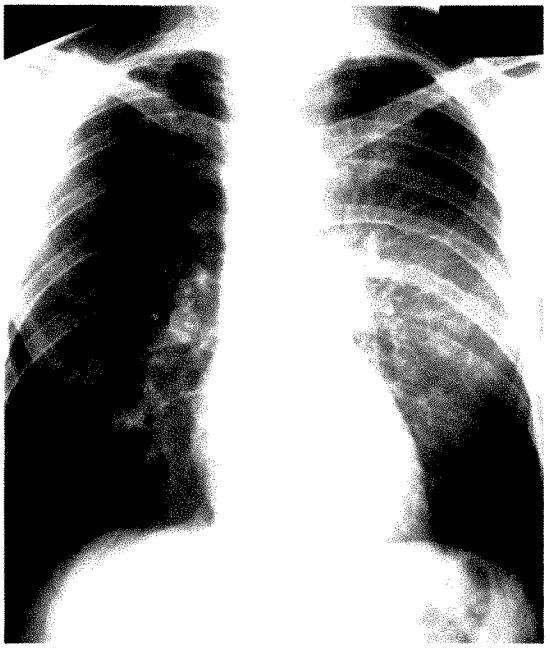


FIG. 13. Arteriovenous fistula involving the left chest wall posteriorly in a 40 year old man. Note scalloped defects of inferior margin of the seventh, of both the inferior and superior margins of the eighth, and of superior margin of the ninth rib on left. Diffuse haziness in this region is due to associated large soft-tissue mass.

rib notching occurs only on the involved side in cases of innominate vein or subclavian vein obstruction on either side. McCord and Bavendam,[16] in 1952, described a case in which long-standing obstruction of the innominate vein was associated with rib notching. In this case, the rib notching on the right was due to dilated and tortuous intercostal veins. In 1958, Dow[6] cited 2 more cases of rib notching due to dilated intercostal veins associated with thrombosis of the subclavian vein or superior vena cava.

Rib notching in such cases is extremely rare, as might be expected, since dilated intercostal veins would not erode the ribs as readily as would dilated, highly pulsatile intercostal arteries. We have not observed rib notching in any case of obstruction of the superior vena cava, innominate, or subclavian vein.

INTERCOSTAL NERVE TUMORS

Neurofibromatosis. In neurofibromatosis, rib notching due to intercostal neurofibromas is commonly encountered (Fig. 14). The rib deformities seen in neurofibromatosis are frequently a part of the generalized bony dysplasia that occurs in this condition. In an occasional case, the involvement of many ribs may suggest the rib notching of coarctation of the aorta. In most cases, however, the associated soft-tissue mass, or masses in the thoracic wall or mediastinum, and other skeletal deformities are visible, and the diagnosis is apparent.

In 11 of 192 cases of neurofibromatosis reviewed at the Mayo Clinic by Hunt,[12] rib erosion was apparent. In 8 cases, multiple ribs were eroded and, in 3, only a single rib was eroded. In each instance associated findings suggested the diagnosis of neurofibromatosis.

Other Neurogenic Tumors. Rib erosion also may occur secondary to other benign

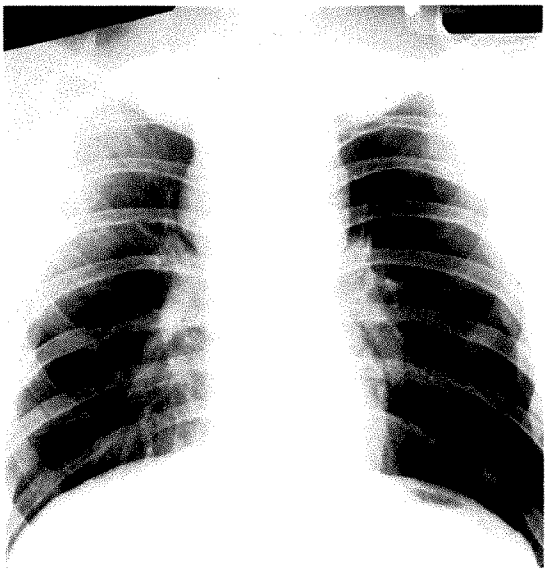


FIG. 14. Bilateral notching of ribs in 22 year old man, similar to that seen in coarctation of aorta. Mediastinal and apical masses and distribution and character of the rib notching, however, are indicative of generalized neurofibromatosis. Hunt, J. C., and Pugh, D. G. Skeletal lesions in neurofibromatosis. *Radiology*, 1961, *76*, 1–19.

intercostal nerve tumors such as schwannomas or neurilemmomas. In these cases, there is usually a single tumor rather than multiple lesions as seen in neurofibromatosis. Generally, only a single rib, therefore, is eroded. In most such cases, the associated tumor mass is visible.

Primary and metastatic tumors of the chest wall produce rib destruction rather than erosion, although erosion may occur early in the growth of these tumors. Such erosion, however, does not usually give rise to the picture of true rib notching due to erosion of the costal grooves.

OTHER REPORTED CAUSES OF RIB NOTCHING

Erosion of the ribs due to dilated intercostal arteries has been reported in cases of arteriosclerosis, hypertension, and other conditions that increase the systemic pulse pressure. In 1937, Laubry and de Balsac[14] reported rib notching in 5 cases, in 2 of aortic valve disease and hypertension, in 1 case of syphilitic aortitis and aortic insufficiency, in 1 of mitral stenosis, and in 1 of valvular heart disease of unknown origin. These authors did not suggest an explanation for the rib notching in these cases. McCord and Bavendam,[16] however, suggested that in some of Laubry and de Balsac's cases, rib erosion may have been due to increased pulsation in the intercostal arteries secondary to an increased pulse pressure.

Dussaillant, Viviani, and Moya,[7] in 1945, reported a case in which rib notching occurred on the left and was presumed due to generalized arteriosclerosis that involved the intercostal arteries.

We have observed no instances of rib notching in these various conditions.

A number of authors have reported "idiopathic" rib notching in cases in which no obvious explanation for the rib notching could be made. Although true rib notching may possibly occur without demonstrable cause, one wonders, if in most such cases, the apparent rib notching is not due to anatomic variation of the ribs themselves rather than to true rib erosion. Thinning of the undersurface of the mid-thoracic ribs near the vertebral column that resembles rib notching is commonly seen and is a normal finding. We have not observed what we would call true rib notching in any patient in whom an underlying cause was not eventually found.

Budenz,[4] in 1950, described irregular cortical thickening that affected the inferior margins of several ribs in patients who had tuberous sclerosis. He suggested that the resulting appearance was similar to the rib notching seen in coarctation of the aorta. The appearance in these cases, however, was more that of irregular cortical thickening than of true rib notching.

Bernstein and associates[2] described erosive rib lesions in 14 of 28 cases of paralytic poliomyelitis. In each instance, the rib erosion was on the superior, posterior aspect of the ribs and was attributed to the continued pressure of the scapula against the ribs. The rib erosion in such cases could hardly be confused with true rib notching which involves the inferior margins of the ribs.

Subperiosteal erosion of the ribs has been reported in cases of hyperparathyroidism by Noetzli and Steinbach.[17] In these cases, the erosion was also on the superior margin of the ribs except when extensive rib changes were present. Such rib erosion is not to be confused with true rib notching and, therefore, this and several of the other conditions just mentioned are not listed in the table.

SUMMARY AND CONCLUSIONS

Rib erosion, or notching of the inferior borders of the ribs, is a roentgenologic sign which may be encountered in various pathologic conditions. It is due most commonly to coarctation of the aorta and to surgical interruption of the subclavian and vertebral arteries for purposes of subclavian artery-pulmonary artery anastomosis in patients with obstruction to pulmonary

blood flow. Rib notching secondary to other lesions is, indeed, rare.

Rib notching seen on thoracic roentgenograms may have important diagnostic implications and, on occasion, may present a difficult diagnostic problem. However, the presence or absence of associated roentgenologic signs and knowledge of the clinical history and physical findings in such patients will permit the radiologist to suggest the correct diagnosis in most cases.

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RIB NOTCHING: ITS MANY CAUSES*

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ONCE thought to be pathognomonic of coarctation of the aorta, notching of the ribs is now known to occur in a variety of other conditions. For the most part, these are related to the components of the intercostal space: the artery, vein, and nerve. Enlargement of any of these structures may result in erosion of the inferior border of the rib. In Table I the causes of rib notching are listed, grouped according to the intercostal structure involved. Each of the conditions enumerated will be separately discussed.

TABLE I

CAUSES OF RIB NOTCHING

- I. Arterial
  - A. Aortic obstruction
    - 1. Coarctation of the aortic arch
    - 2. Thrombosis of the abdominal aorta
  - B. Subclavian artery obstruction
    - 1. Blalock-Taussig operation
    - 2. "Pulseless disease"
  - C. Widened arterial pulse pressure (?)
  - D. Decreased pulmonary blood supply
    - 1. Tetralogy of Fallot
    - 2. Pulmonary atresia (pseudotruncus)
    - 3. Ebstein's malformation
    - 4. Pulmonary valve stenosis
    - 5. Unilateral absence of the pulmonary artery
    - 6. Pulmonary emphysema
- II. Venous
  - A. Superior vena cava obstruction
- III. Arteriovenous
  - A. Pulmonary arteriovenous fistula
  - B. Intercostal arteriovenous fistula
- IV. Neurogenic
  - A. Intercostal neurinoma
- V. Osseous
  - A. Hyperparathyroidism
- VI. Idiopathic
- VII. Normal

COARCTATION OF THE AORTA

Following the first roentgen description of notching of the ribs in coarctation of the aorta by Rösler[35] in 1928 (independently reported the next year by Railsback and Dock[33]), the sign was for many years widely accepted as diagnostic of this disease.

Numerous studies have demonstrated the mechanism of the production of notching in coarctation.[3,6,34] The collateral flow bypassing the aortic constriction arises almost exclusively from the subclavian arteries. Anastomoses between branches of the subclavian arteries and the aortic intercostal arteries conduct blood to the aorta distal to the coarctation. The blood flows through 3 main pathways (Fig. 1): (1) the scapular group, composed of the transverse scapular, transverse cervical, and superficial cervical arteries from the thyrocervical trunk, and the subscapular branch of the axillary artery; (2) the external thoracic group, consisting of the short and long thoracic branches of the axillary artery; and (3) the internal thoracic group, comprising the two internal mammary arteries and their anterior intercostal branches.[28]

The flow through the intercostal arteries in coarctation of the aorta is, of course, opposite to the normal direction of flow. The large volume of blood causes dilatation, tortuosity, and increased pulsation of the intercostal arteries. The intercostal arteries may approach the subclavian artery in size,[37] reaching aneurysmal proportions in about 8 per cent of cases.[12] Proximity of the artery to the inferior border of the posterior rib results in gradual erosion of the bone.

When a patent ductus arteriosus enters the aorta distal to the coarctation, rib notching is ordinarily not seen. In this event, blood is supplied to the lower portion of the body from the pulmonary circulation

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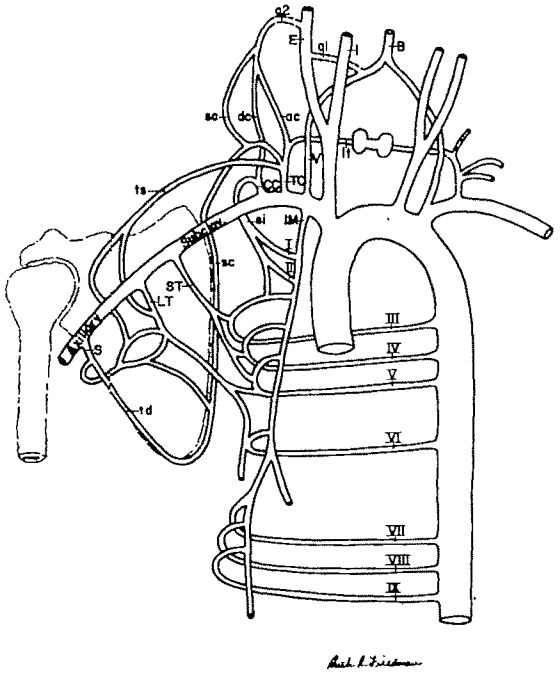


FIG. 1. Collateral arterial pathways in cervicothoracic region. Capital letters designate major vessels; small letters indicate secondary branches; Roman numerals refer to intercostal arteries.

B—Basilar	a1—External carotid—vertebral anastomosis
CC—Costocervical trunk	a2—External carotid—cervical anastomosis
E—External carotid	ac—Ascending cervical
I—Internal carotid	dc—Deep cervical
IM—Internal mammary	it—Inferior thyroid
LT—Long (lateral) thoracic	sc—Superficial cervical
S—Subscapular	si—Superior intercostal
ST—Short (superior) thoracic	td—Thoracodorsal
TC—Thyrocervical trunk	ts—Transverse scapular
V—Vertebral	

through the open ductus. Thus, the intercostal arteries do not become important collateral pathways.

In coarctation, the notching is nearly always bilateral, but unilateral notching does occur[3] and is readily explained on a developmental basis (Fig. 2). If the coarctation develops distal to the left subclavian artery, bilateral notching occurs. If the coarctation develops proximal to this vessel or involves its orifice, as it does in about 2 per cent of cases,[6] the notching is confined to the right side. An aberrant right subclavian artery arising as the last vessel from the aortic arch is found in 1 per cent of coarctations.[6] Left-sided notching occurs when this vessel originates distal to the coarctation. In these special circumstances only the subclavian artery arising proximal to the aortic obstruction serves as an effective collateral pathway to the intercostal

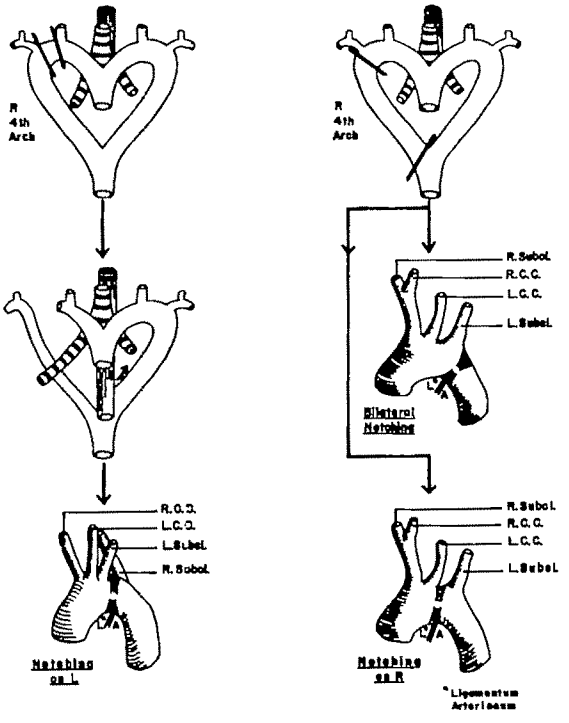


FIG. 2. Embryologic explanation for unilateral rib notching in coarctation of the aorta. *Top right and left* show the double fourth arches normally present in early fetal life. The segment between the heavy black lines subsequently disappears. *Middle left.* Formation of aberrant right subclavian artery. *Lower left.* Aortic coarctation proximal to the aberrant right subclavian artery. This results in unilateral rib notching on the left. *Middle right.* The usual site of coarctation which produces bilateral rib notching. *Lower right.* The coarctation lies proximal to the left subclavian artery, resulting in rib notching on the right. (Reproduced with permission from Williams & Wilkins Co., Publisher.)

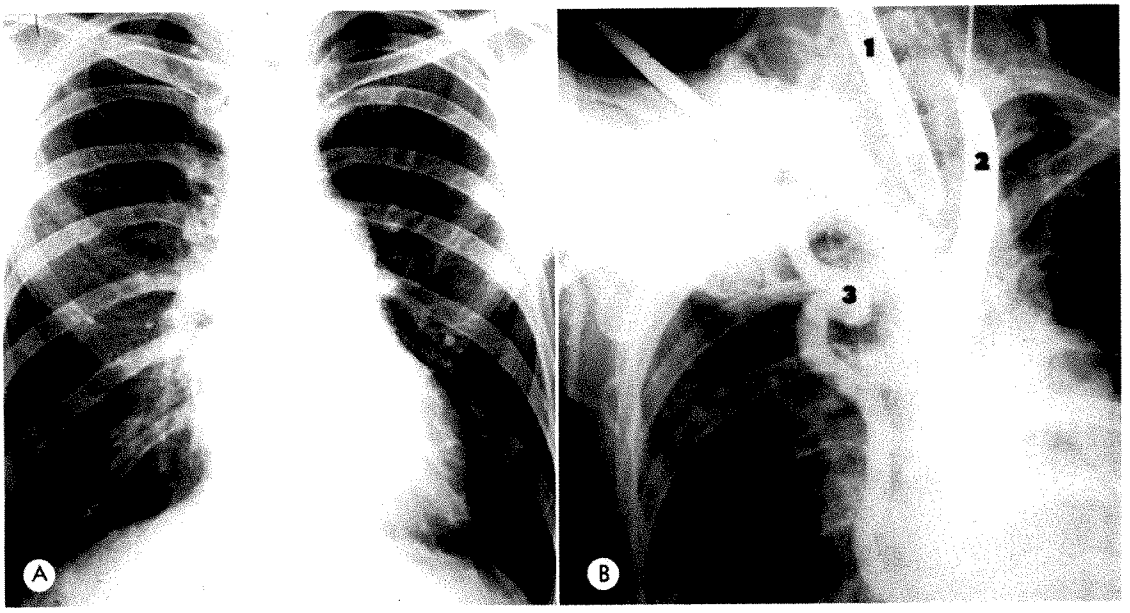


Fig. 3. Coarctation proximal to the left subclavian artery. (*A*) There is notching of the right fifth to seventh ribs. (*B*) Retrograde left carotid arteriogram. The coarctation is obscured by the spinal shadow. 1. Tracheotomy tube. 2. Left common carotid artery. 3. Greatly enlarged right internal mammary artery. The left internal mammary artery is diminutive. The right brachiocephalic arteries have filled from the aorta. Their branches, partially obscured by the patient's hand, are considerably enlarged. The intercostal arteries have not yet filled.

arteries, so that the rib notching is confined to that side.

Figure 3*A* shows marked notching of the right fifth, sixth, and seventh ribs. The left ribs are normal. On the retrograde left common carotid arteriogram (Fig. 3*B*), the right internal mammary artery is enormously dilated and the cervicoscapular branches of the right subclavian artery are enlarged. The left internal mammary artery is quite small. At operation, the aorta was found to be occluded between the left common carotid and the left subclavian arteries. Figure 4*A* shows notching confined to the left sixth and seventh ribs. The upper esophagus (Fig. 4*B*) shows the typical posterior imprint of an anomalous right subclavian artery.[14]

Unilateral rib notching, however, is not always so simply explained. We have seen 2 examples of unilateral notching in which aortography showed both subclavian arteries arising proximal to the coarctation in the usual manner.

Rib notching is present in over 75 per cent of adults with coarctation,[34] but is said to occur very rarely in early childhood.[8] It is surprising, therefore, that in a recent article definite notching was reported in 13 per cent of infants under 1 year of age with coarctation.[26] Our own experience indicates that rib notching occurs fairly often in children under 12 but is quite rare in infants. Unquestionably, the time factor plays a significant role in the development of rib notching. However, the severity of the aortic obstruction is probably even more important.[32] Notching is more apt to appear early in life if the occlusion is marked and may never occur at all with lesser degrees of constriction. Yet, we have encountered several asymptomatic patients in the seventh and eighth decades with coarctation and striking rib notching. Presumably they had only mild or moderate coarctation and the duration accounted for the severity of the notching.

Notching of the first two ribs is quite rare in coarctation of the aorta. The upper two

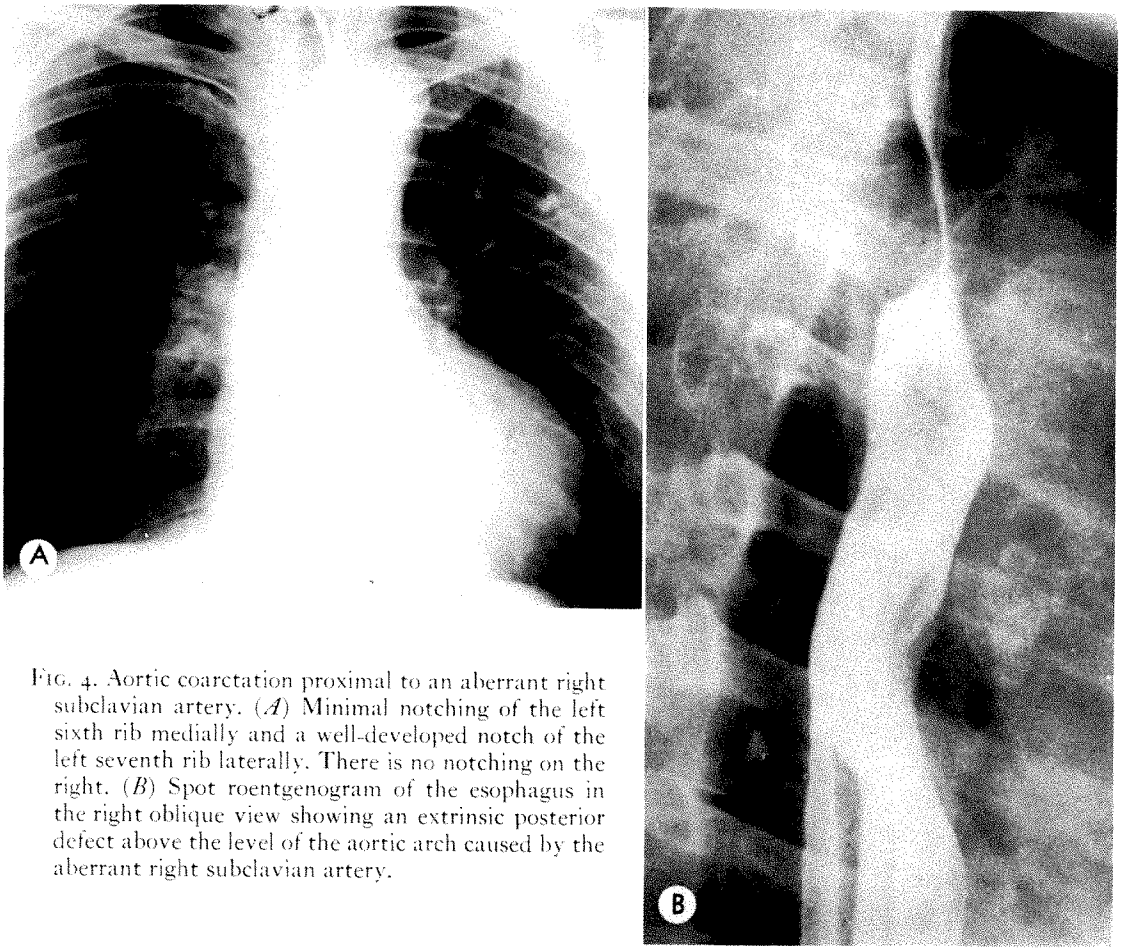


FIG. 4. Aortic coarctation proximal to an aberrant right subclavian artery. (*A*) Minimal notching of the left sixth rib medially and a well-developed notch of the left seventh rib laterally. There is no notching on the right. (*B*) Spot roentgenogram of the esophagus in the right oblique view showing an extrinsic posterior defect above the level of the aortic arch caused by the aberrant right subclavian artery.

intercostal arteries nearly always arise from the superior intercostal artery, a branch of the costocervical artery, rather than from the aorta. They do not convey blood *directly* to the aorta below the obstruction, as do the other intercostal arteries. Furthermore, they pass obliquely toward the subcostal groove, lying below the more horizontal intercostal nerve and farther from the corresponding rib.[28]

The tenth and eleventh posterior intercostal arteries do not anastomose with the anterior intercostal branches of the internal mammary arteries, but instead join the inferior and lateral epigastric branches of the external iliac arteries.[28] The twelfth intercostal (or subcostal) artery is essentially a lumbar artery. The lower three intercostal arteries lie at some distance from the aortic obstruction and probably participate little in the collateral flow. In experimental acute occlusion of the aortic isthmus in 3 dogs, we noted that contrast medium in the distal aorta flowed outward into the lower intercostal arteries.[16] For all these reasons the last three ribs are not notched in coarctation of the aorta.

Notching from coarctation, then, is rarely seen above the third or below the ninth rib. This is not to say that all the ribs between are involved in an individual case. In fact, this is seldom so and in most cases the notching is asymmetric and confined to only a few ribs on either side. The notches on the affected rib may be single or multiple, shallow or deep, broad or narrow. The location of the indentations is quite variable from case to case and from one rib to the next

(Fig. 4*A*). The anterior rib segments are not affected because the anterior intercostal arteries, branches of the internal mammary arteries, are bifid and do not run in a subcostal groove as do the aortic (posterior) intercostal arteries. The superior aspect of a rib may be notched if the tortuosity of the superjacent posterior intercostal artery is so marked that it makes contact with the rib below.[38]

LOWER AORTIC OBSTRUCTION

Obstruction of the lower thoracic or abdominal aorta also involves the intercostal arteries in the collateral flow. This occurs in atypically located coarctation[40] and in aortic thrombosis. Figure 5 is a thoracic aortogram in a patient with thrombosis of the lower abdominal aorta, demonstrating the dilated tortuous lower intercostal arteries. These collaterals convey blood from the aorta to the lower extremities, bypassing the obstruction, and the intercostal flow is in a direction opposite to that in coarctation. Although the lower ribs are spared in coarctation, the extreme dilatation and tortuosity of the intercostal arteries which occur in obstruction of the lower aorta may produce notching, which was present but not well developed in this instance.

SUBCLAVIAN OBSTRUCTION FOLLOWING THE BLALOCK-TAUSSIG OPERATION

In the Blalock-Taussig procedure for congenital cyanotic heart disease, the subclavian artery is divided and its proximal end is anastomosed with the pulmonary artery on the same side, increasing the blood flow to the oligemic lungs. Following the operation, a number of collateral pathways to the arm distal to the ligated subclavian artery are available* (Fig. 1). However, in order to obtain sufficient mobilization for the anastomosis, some or all of the major branches of the subclavian artery may be ligated, blocking many of these potential pathways.[9,21] In this event, the posterior intercostals may carry the bulk of the col-



FIG. 5. Thrombosis of abdominal aorta. The thoracic aortogram shows dilatation and tortuosity of the eighth, ninth, and tenth intercostal arteries, which supply blood to the lower part of the body via anastomoses with arteries of the abdominal wall. On the plain roentgenogram a small notch was visible in the left tenth rib medially.

* 1. Aorta →posterior intercostal arteries →subscapular and long and short thoracic branches of axillary artery.

2. Proximal subclavian artery →thyrocervical →transverse scapular, transverse cervical, and superficial cervical arteries.

3. Proximal subclavian artery →costocervical →superior intercostal →internal mammary →intercostal arteries →subscapular and thoracic branches of axillary artery.

4. Proximal subclavian artery →unnamed arteries in the axilla.

5. Contralateral vertebral artery →proximal basilar →(retrograde) ipsilateral vertebral artery.

6. Contralateral subclavian artery →thyrocervical and costocervical branches →ipsilateral branches.

7. External carotid artery →occipital →vertebral, thyrocervical, or costocervical branches.[28]

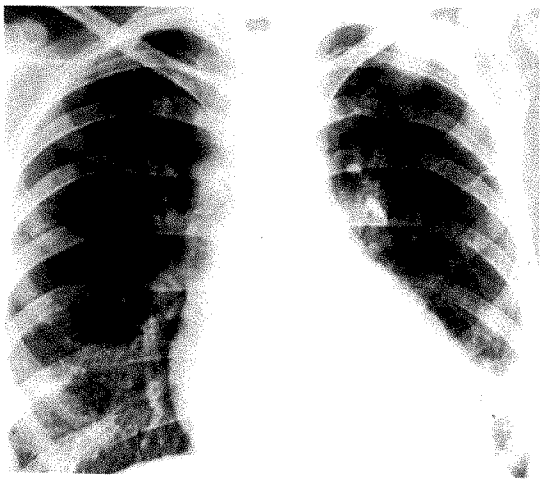


FIG. 6. Patient had a Blalock-Taussig operation for tetralogy of Fallot. Notching is seen in the third to fifth ribs on the left, the side of the operation. A roentgenogram made 2 years earlier, at the time of the operation, showed no notching.

lateral flow through their communications with the thoracic and subscapular branches of the axillary artery.[9,31]

Kent,[21] in 1953, encountered 2 cases of ipsilateral rib notching following the Blalock-Taussig operation. In one, angiography demonstrated the dilated intercostal arteries responsible for the notching. The following year Levin and Rigler[24] reported 8 additional cases. They stated that notching occurred only if the anastomosis became occluded or if, for technical reasons, it could not be completed after the subclavian artery had been ligated. Peterson found that 17 of 100 operated patients had developed ipsilateral notching. However, 14 of the 17 had evidence of a functioning anastomosis.[30]

Obviously, then, closure of the anastomosis is not essential for rib notching to appear. Nevertheless, notching occurs in a higher percentage of patients in whom the anastomosis has become occluded. In these instances, it is presumed that the thrombus extends to include the orifices of some of the major branches of the subclavian, causing more of the collateral blood to take the intercostal route to the arm.

Figure 6 demonstrates well-developed notches in the left third, fourth, and fifth ribs in a young woman who had a left Blalock-Taussig operation for tetralogy of Fallot 2 years earlier. There had been no notching before the operation. The anastomosis was thought to be occluded because cyanosis recurred and the bruit disappeared.

PULSELESS DISEASE

The term "pulseless disease" has been applied to occlusion of two or more brachycephalic arteries at or near their origin, resulting in absent pulses in their corresponding distributions.[11] Underlying causes include arteriosclerosis obliterans, luetic aortitis (with or without aneurysm), arteritis of the Takayasu type, and post-traumatic thrombosis.[36]

We have found only 1 previously reported case of rib notching accompanying this condition.[32] This was in a young Japanese woman with Takayasu's disease. The mechanism of the notching is the same as that following the Blalock-Taussig operation: collateral circulation to the arm via the intercostal arteries.

In the so-called "subclavian steal" syndrome, an important route of collateral flow is demonstrated which may supersede the intercostal pathway in obstruction of the proximal subclavian artery. As shown by injection of contrast medium, blood from the contralateral vertebral artery bypasses the basilar artery and flows retrograde into the ipsilateral vertebral artery, entering the subclavian artery distal to the obstruction. Thus, the subclavian artery "steals" blood intended for the brain. This and the many other patent cervical connections[31] (Fig. 1) sidetrack the blood from the intercostal route, probably explaining why rib notching so seldom occurs in isolated occlusion near the orifice of a subclavian artery. Figure 7, *A* and *B*, shows left rib notching in a patient with severe calcific atherosclerotic changes at the origins of all the brachycephalic arteries. Pulses were absent in both arms and both sides of the neck.

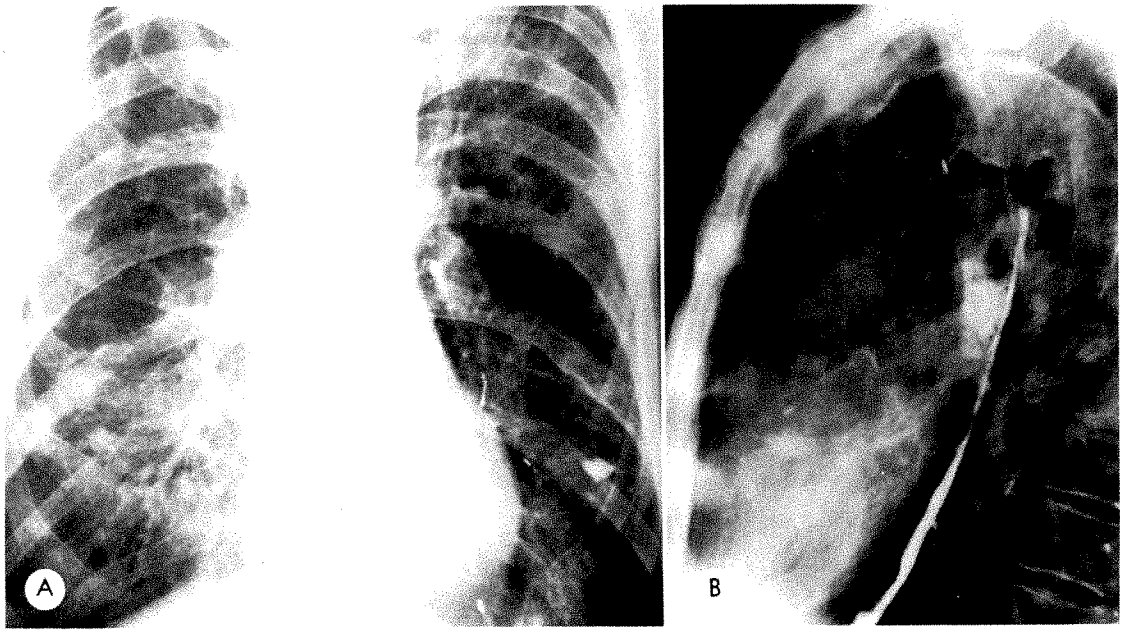


FIG. 7. "Pulseless disease." (*A*) There is definite notching of the left eighth rib. (*B*) Extensive calcification is present in the wall of the aorta and brachycephalic arteries. A calcified thrombus is thought to be present in one of the great vessels, just anterior to the barium-filled upper esophagus. A small amount of barium has been aspirated. Pulses were absent in both arms and both sides of the neck, but the femoral pulses were strong. The patient expired and autopsy was not performed.

WIDENED ARTERIAL PULSE PRESSURE

Laubry and Heim de Balsac,[23] in 1937, reported rib notching "in older people with striking aortic changes, often associated with hypertension." They also encountered it in syphilitic aortitis with aortic insufficiency. There has been one further case report of notching in arteriosclerosis, but this was not proved.[19] It has been postulated that the rib erosion in these cases was related to an increase in the pulse pressure.[27] However, in view of the large number of cases of hypertension with generalized arteriosclerosis, the incidence of notching due to this mechanism must be quite rare, if it does indeed occur.

In reviewing the chest roentgenograms of 100 unselected patients who at autopsy showed marked generalized atherosclerosis without large vessel occlusion, we found 2 examples of mild to moderate notching of one and three ribs, respectively. However, as will be shown later, similar degrees of notching can be seen with equal frequency in normal individuals.

DECREASED PULMONARY BLOOD SUPPLY

From a hemodynamic standpoint, it is possible for rib notching to develop in any congenital cardiovascular condition associated with decreased pulmonary arterial blood flow. The development of notching depends upon the presence of an extensive collateral blood supply to the oligemic lungs from the systemic circulation via the intercostal arteries. This systemic collateral circulation may become so well developed that the lungs actually appear hyperemic (Fig. 8).[22] There are multiple routes by which the systemic blood may reach the lungs in these cases. The aortic intercostal arteries are intimately involved in these pathways.* They may enlarge sufficiently to cause rib notching.

Notching has been reported in the tetralogy of Fallot (prior to operation),[4,9] uni-

* 1. Proximal descending aorta or aortic intercostal arteries →bronchial arteries (usually two on left, one on right).

2. Aortic intercostal arteries→internal mammary, phrenic, and pericardial arteries→bronchial branches.

3. Aorta, subclavian, and phrenic artery→anomalous branches into lung.[42]

4. Aortic intercostal arteries→perforating pulmonary branches.[1]

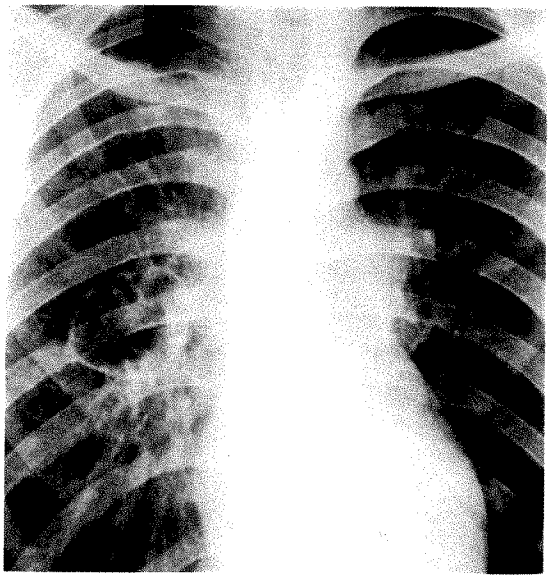


FIG. 8. Pulmonary valvular stenosis with patent foramen ovale. There is notching of the right fourth to sixth ribs. The hyperemia of the right lung may be attributable to extensive collateral circulation. The reason for the unilateral distribution of the notching is not clear. The thin-walled cavity in the right lung proved to be a bronchogenic cyst.

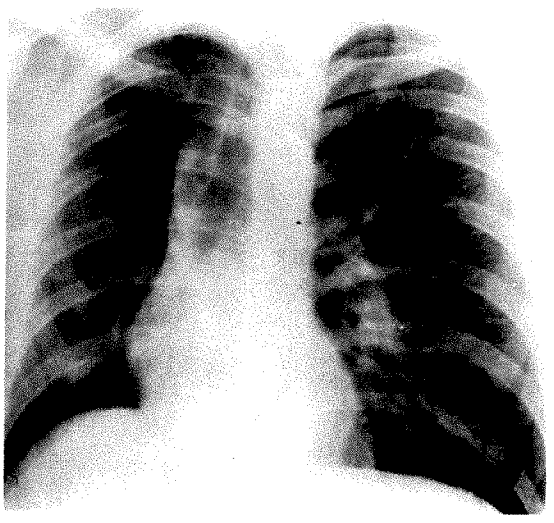


FIG. 9. Congenital absence of the right pulmonary artery. The heart and mediastinal structures are shifted into the right thorax, which is smaller than the left. The left lung is hyperemic because it is receiving all the blood from the right ventricle. Notching (arrow) is present in the right fifth and sixth ribs and is the result of collateral flow to the right lung via the intercostal arteries. There was no evidence of heart disease. Unproved. (Reproduced with permission from W. B. Saunders Co., Publisher.)

lateral absence of the pulmonary artery,[13] pulmonary artery atresia (pseudotruncus),[9] and Ebstein's malformation.[9] Curiously, even in patients with bilateral pulmonary oligemia, the notching has been invariably unilateral. This has also been true in our own cases. We have no explanation for this.*

Figure 8 shows a patient with pulmonary valve stenosis and patent foramen ovale. There is notching of the fourth, fifth, and sixth right ribs. In Figure 9, a patient with congenital absence of the right pulmonary artery, notching of the right fifth and sixth ribs is shown. Figure 10 is an example of rib notching on the right in a patient with tetralogy of Fallot and absent right pulmonary artery. The small notch in the left sixth rib first appeared one year after a left Blalock-Taussig operation. Thus, there is notching of different etiology on the two sides.

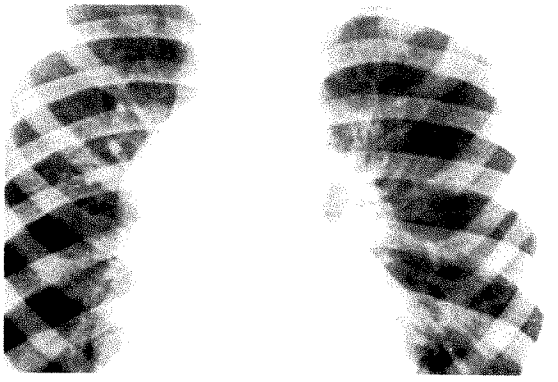


FIG. 10. Tetralogy of Fallot with absent right pulmonary artery. Slight notching of the left sixth rib appeared 1 year after an unsuccessful left Blalock-Taussig operation. The mild notching of the right sixth and seventh ribs was present prior to this operation. Subsequent right thoracotomy showed absence of the right pulmonary artery. The patient thus illustrates two different mechanisms of rib notching.

* Acquired unilateral notching has been reported recently in pulmonary emphysema,[18] from this same mechanism. One of our cases, previously considered to be idiopathic notching, is probably an example of this.

VENOUS OBSTRUCTION

Although one might not expect soft nonpulsatile veins to erode bone, striking costal notching has been encountered in chronic obstruction of the superior vena cava.[2,27] Fibrosing mediastinitis has so far been the chief underlying cause in these cases, probably because of its prolonged course. If the obstruction lies entirely above the entrance of the azygos vein, much of the blood from the upper portion of the body reaches the patent segment of the superior cava through the azygos vein. If the obstruction incorporates or lies below the mouth of the azygos vein, the flow is principally toward the inferior vena cava. In either of these circumstances, the intercostal veins carry a considerable excess of blood and may produce costal erosion.[15]

Figure 11*A* shows, in addition to fullness of the superior mediastinum, bilateral notching of the ribs. The right brachial venogram (Fig. 11*B*) shows obstruction of the superior vena cava below the azygos vein entry. A markedly enlarged and tortuous right intercostal vein projects into one of the notches. We have seen 2 additional examples of venous rib erosion from chronic fibrosing mediastinitis.

ARTERIOVENOUS FISTULA

Rib notching in pulmonary arteriovenous fistula is not common.[44] In one autopsied case, the pleural space over the right middle and lower lobes was obliterated and numerous large tortuous vessels extended from the chest wall to the lung, fed by three intercostal arteries.[39] Traumatic and congenital intercostal arteriovenous fistulas may also be associated with notching of the adjacent ribs.[17,25] Notching has not been encountered with simple cavernous hemangioma of the chest wall.[41]

Figure 12, *A* and *B*, is an example of rib notching in a patient with innumerable pulmonary arteriovenous fistulas and hereditary familial telangiectasis (Osler-Rendu-Weber syndrome).

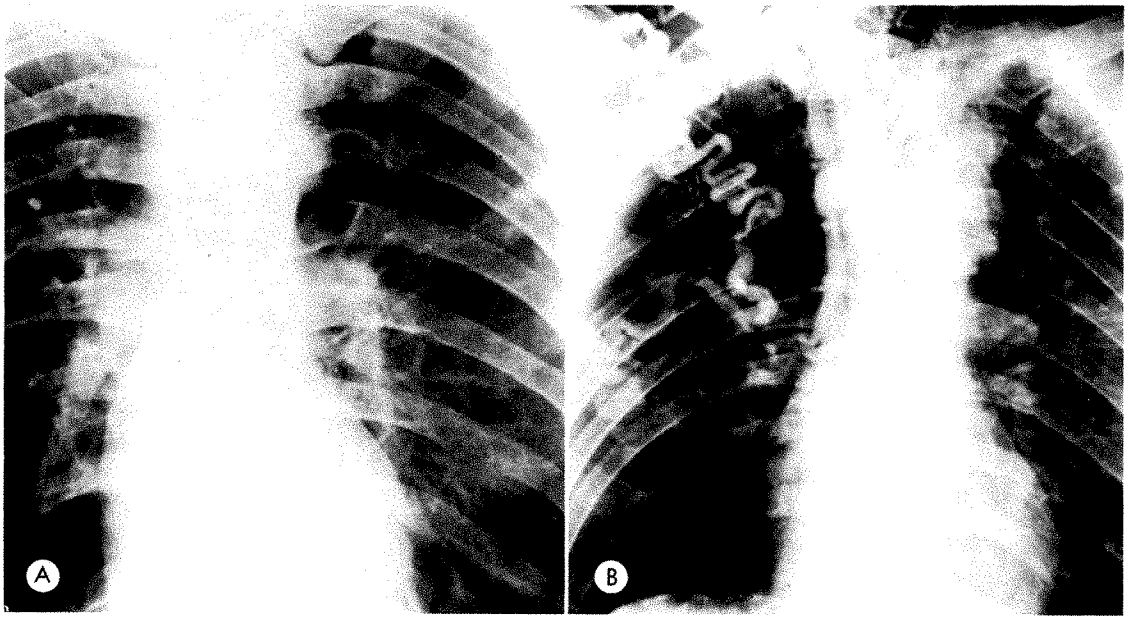


Fig. 11. Superior vena caval obstruction secondary to chronic fibrosing mediastinitis, probably of histoplasmic origin. There is notching of the left seventh and eighth and right fifth to seventh ribs. (*A*) Plain roentgenogram. The prominence in the region of the aortic knob represents the enlarged superior accessory hemiazygos vein. (*B*) Right brachial venogram revealing numerous collaterals in the neck and mediastinum. The superior vena cava is occluded below the entry of the azygos vein, which shows retrograde flow. A very large tortuous intercostal vein conforms to some of the rib notches.

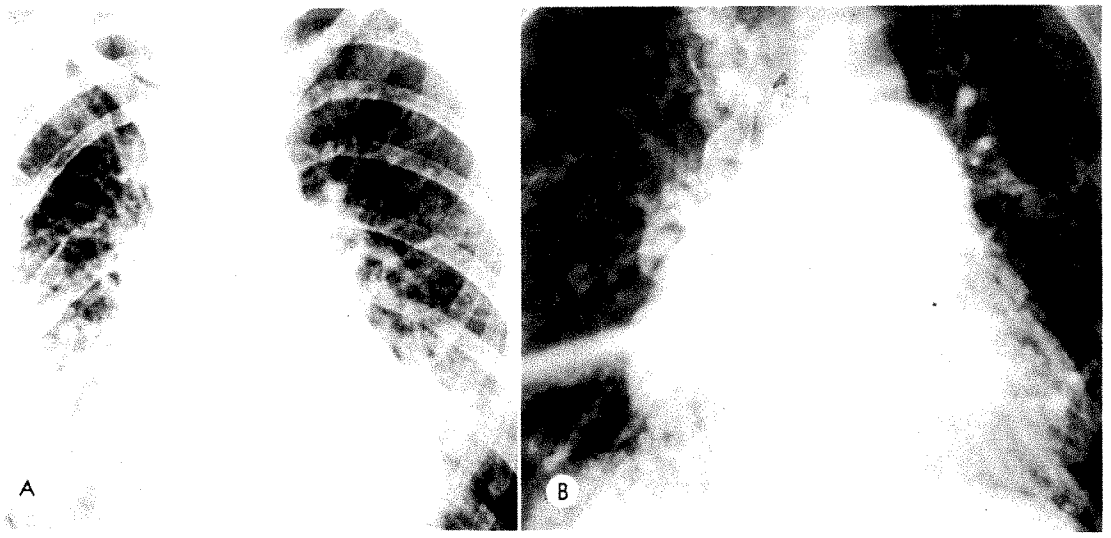


FIG. 12. Extensive bilateral pulmonary arteriovenous malformation in a patient with Osler-Rendu-Weber's syndrome. (*A*) Plain roentgenogram. There is notching of the right fifth and seventh ribs. The sixth rib has been resected. The heart is considerably enlarged. (*B*) There are innumerable small arteriovenous fistulas visible on the angiocardiogram. The density in the right lower thorax is an artefact.

NEUROGENIC TUMOR

Rib deformity is commonly encountered in patients with solitary or multiple intercostal or spinal nerve tumors. Pressure from the tumor or congenital maldevelopment related to it may result in attenuation, gross irregularity, or displacement of the affected rib.[19] However, true notching by intercostal neurinoma does occur, particularly in neurofibromatosis. Many ribs may be affected, simulating the rib notching seen in coarctation.[18] In our own cases of notching from neurofibroma, one or more extrapleural masses were roentgenographically visible somewhere in the thorax. Similar masses have not been encountered in other conditions associated with notching. Figures 13 and 14, *A* and *B*, show rib notching in 3 patients with Von Recklinghausen's disease.

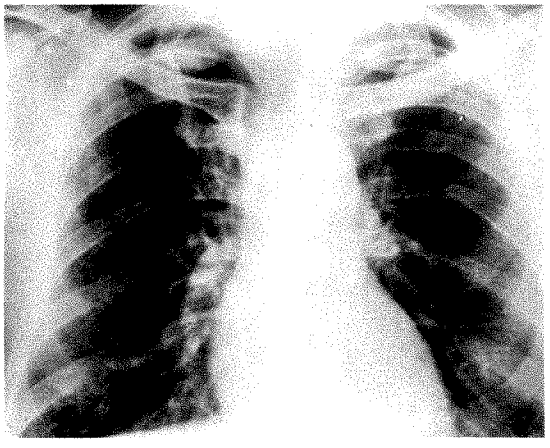


FIG. 13. Neurofibromatosis. There is notching of the right fourth to seventh and the left sixth to eighth ribs. The two masses protruding into the pulmonary apices probably represent extrapleural extension of intercostal neurofibromas.

OSSEOUS CONDITIONS

Several references are made in the literature to "pseudo-rib notching" in tuberous sclerosis.[24,43] These stem from a report in which notching of several ribs was described in a case purported to be tuberous sclerosis.[7] However, the rib changes appear to represent irregular cortical thickening rather than notching and the clinical findings seem more in keeping with Gardner's syndrome, since there were multiple colonic polyps, exostoses of the skull, and multiple subcutaneous xanthomas, fibromas, and sebaceous cysts. The attenuation of the third to eighth posterior ribs (from scapular pressure) en-

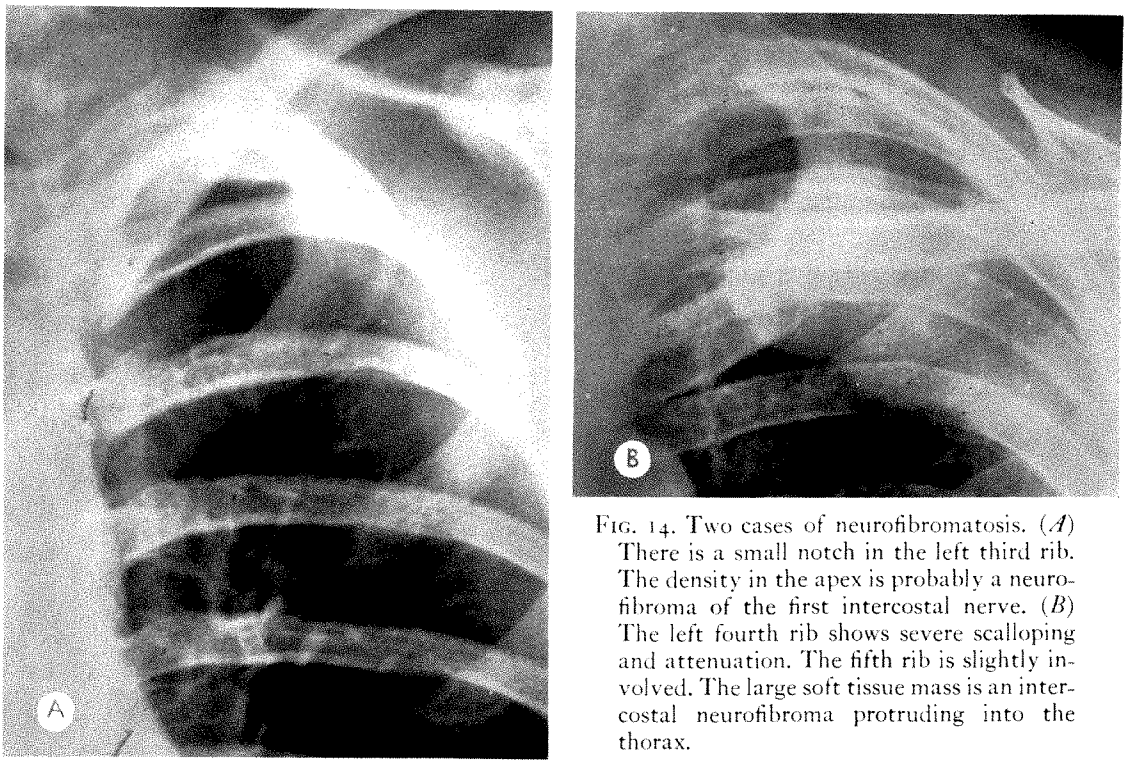


Fig. 14. Two cases of neurofibromatosis. (*A*) There is a small notch in the left third rib. The density in the apex is probably a neurofibroma of the first intercostal nerve. (*B*) The left fourth rib shows severe scalloping and attenuation. The fifth rib is slightly involved. The large soft tissue mass is an intercostal neurofibroma protruding into the thorax.

countered in paralytic poliomyelitis does not resemble rib notching.[5]

In hyperparathyroidism, subperiosteal erosion of bone, particularly in the phalanges and tibias, is a common and specific feature. Similar involvement of the ribs may occur, usually on their superior aspect.[29] However, we have encountered one example of hyperparathyroidism in which the inferior rib surfaces were affected, giving the appearance of notching (Fig. 15).

IDIOPATHIC NOTCHING

Occasional examples of unequivocal rib notching have been encountered in which thorough investigation has failed to reveal any of the causes mentioned above. We have seen one such case with marked bilateral notching. Lesser degrees of notching are encountered fairly often in apparently healthy individuals. One of us (B.F.) has reviewed 1,000 survey chest roentgenograms for rib notching and encountered it in mild to moderate degree with surprising frequency (Table II and Fig. 16).

DISCUSSION

Our list of causes of rib notching, while relatively long, is probably not complete. One can predict with reasonable assurance that notching will eventually be demonstrated in such conditions as coarctation or thrombotic occlusion of a main pulmonary artery, congenital absence or chronic obstruction of the inferior vena cava, bronchiectasis with hypertrophy of the bronchial arteries, and pulmonary oligemia associated with cardiovascular anomalies other than those cited earlier.

It is easy to overlook minor degrees of rib notching on the roentgenogram. Conversely, one must guard against over-reading them. In view of the frequency of minor and even moderate indentations of the ribs in normal individuals, it is sometimes difficult to be certain of pathologic notching. In this connection, the location of the indentation on the rib has been helpful. While abnormal notching is more difficult to recognize medially (because of the greater normal variation here), it is seldom limited to



Fig. 15. Hyperparathyroidism. The left sixth to eighth ribs are notched, probably as a result of subperiosteal resorption. The inner end of the left clavicle shows irregular destruction.

this region. In most cases, true notching is confined to the *middle* third of the posterior ribs. Similarly, multiplicity of rib involvement strongly favors *bona fide* notching. Yet we have seen a fair number of examples of pathologic notching in which only one or two ribs were involved. Shallow, wavy, elongated scalloping is just as reliable from the diagnostic standpoint as deeper, narrower, solitary notches. Despite these rules, we have seen a few cases in which rib notching was equivocal.

Table II

Incidence of Rib Notching in 1,000 Survey Chest Roentgenograms*

	Per Cent
Medial half of posterior ribs	
Mild	19.0
Moderate	2.2
Lateral half of posterior ribs	
Mild	5.0
Moderate	1.4

* One or more ribs affected.



Fig. 16. Normal patient with mild rib notching. The left eighth and ninth ribs are involved.

Little has been written about the length of time it takes for notching to appear and, after correction of the cause, to disappear. In coarctation of the aorta, the notching is occasionally present in early infancy[26] and may regress within a year after surgical correction.[20] Notching after the Blalock-Taussig operation appears, on the average, about 38 months after the procedure, but has been seen as early as 6 months.[21,24,30] We have little to add to this meager information. It seems clear that in the various conditions enumerated the duration as well as the severity of the causative mechanism is important for the production of notching.

Familiarity with the causes and mechanisms of rib notching is of practical value.

The sign is a frequent one and its implications are important. The recognition of this sign will often enable the roentgenologist to direct the further work-up of the patient toward a definitive diagnosis.

SUMMARY

Notching of the ribs is now known to be caused by a variety of conditions. These are related, for the most part, to enlargement of one of the three components in the subcostal grooves, the intercostal arteries, veins, and nerves. The different mechanisms of arterial rib notching are discussed and the collateral pathways are described. The many causes of rib notching are classified and examples of most of them are illustrated.

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We are deeply indebted to the following radiologists for their kind permission to use several of the roentgenograms reproduced in this report: Drs. John A. Campbell, Figure 3, *A* and *B* and Figure 4, *A* and *B*; Lee L. Rosenberg, Figure 8; Warner A. Peck, Figure 11, *A* and *B*; and Russell H. Morgan, Figure 12, *A* and *B*.

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UNUSUAL FORAMINA, PSEUDOFORAMINA, AND DEVELOPMENTAL DEFECTS OF BONE*

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ALL roentgen findings can be classed as "normal" or "abnormal." Ordinarily, for roentgenologists, this differentiation is a comparatively simple matter; however, there are occasions when this decision defies immediate solution. Normal bone and joint structure is learned by every radiologist early in his career, although certain variations of normal may not be as well known.

This report deals with some roentgen findings in bone which are "unusual" in the sense that they occur only in a small minority of the population, but are "normal" in the sense that they are found in healthy people; they concern (1) foramina, (2) pseudoforamina, and (3) developmental defects.

I. FORAMINA

STERNAL FORAMEN

Most congenital sternal anomalies, being asymptomatic, are discovered only by accident when the sternum is incidentally visualized on a roentgenogram taken for some other purpose. It is perhaps for this reason that developmental abnormalities of the sternum are neither as frequently recognized nor as often reported as are congenital anomalies elsewhere in the chest. A rather extensive search throughout the radiologic literature has failed to disclose any description of the rare condition of sternal foramen. This entity is briefly mentioned by Köhler and Zimmer,[16] who state that they have personally seen 4 such cases.

Figure 1 shows a sternal foramen, noticed as an incidental finding during an examination of the esophagus in an essentially healthy person.

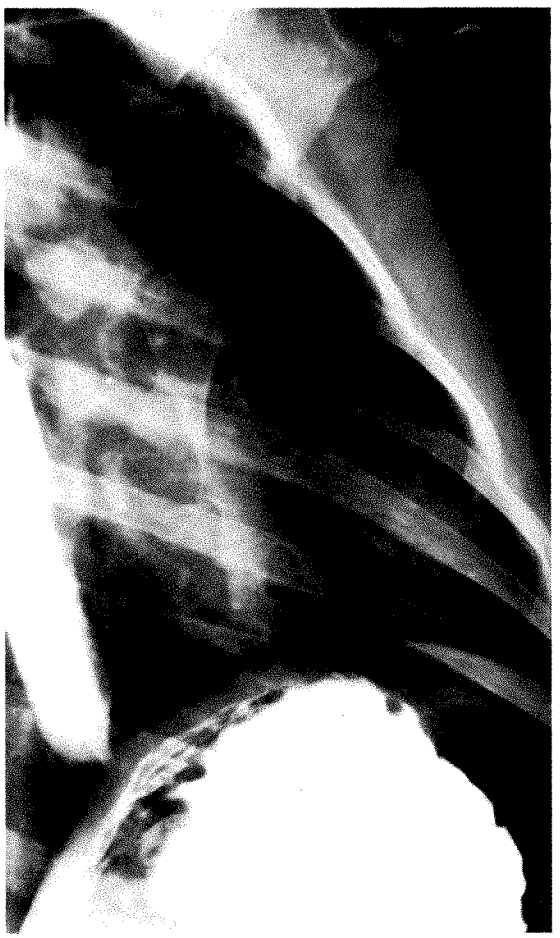


FIG. 1. *Sternal Foramen.* The defect is well seen in the lower portion of the corpus sterni. Barium is present in the esophagus and stomach.

Despite its simplicity of appearance in the adult, the sternum evolves during fetal and neonatal life in a rather complex manner.[26] According to Gray's Anatomy,[10] the sternum originally consists of two parallel cartilaginous bars, each connected with cartilaginous ribs on both sides of the mid-

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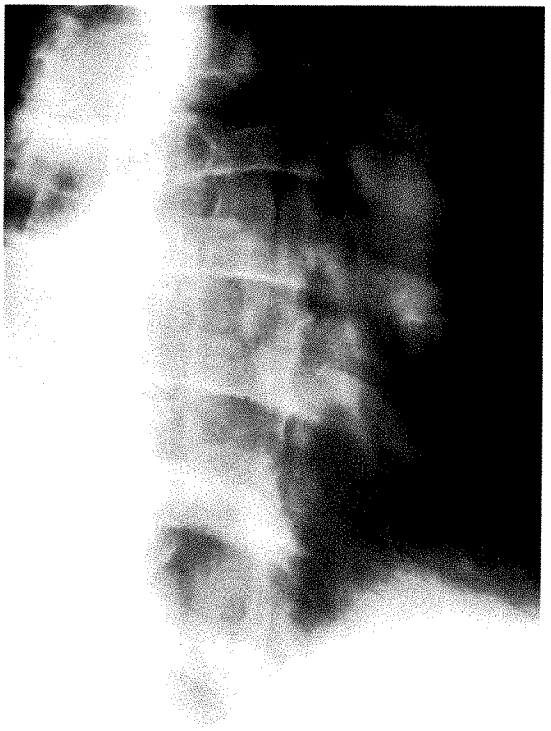


FIG. 2. *Xiphoid Foramen.* This well circumscribed defect in the xiphoid process of the sternum is more common than the sternal foramen. See Figure 1.

line. By the early second trimester of gestation, these two bars fuse with each other; failure of fusion results in a partial or complete vertical fissure. The single cartilaginous sternum then proceeds to develop 6 primary ossification centers: 1 for the manubrium, 4 for the body, and one for the xiphoid process. These appear first in the manubrium in the sixth fetal month, and develop in a cranial-caudal direction until the xiphoid center forms in the fifth to eighteenth year of life. In addition, two episternal centers sometimes occur.[3] Occasionally, some of the segments of the sternal body arise from several ossification centers. The second, third, and fourth segments are not uncommonly formed from two centers placed laterally to each other. The irregular midline union of these explains the rare occurrence of the sternal foramen.[30] For the sake of completeness, it should be noted that fusion of the ossified sternal segments begins at puberty and proceeds in a caudal cranial direction; complete union into a single bone does not occur until the twenty-fifth year of life.

The sternal foramen is of varying size and form, and although it is said to contain a massive vascular bundle during fetal life,[17] it is filled with hyaline cartilage in the adult.[8] Although a foramen may also occur in the xiphoid process, the true sternal foramen occurs only at the junction of the third and fourth segments of the sternal body, at the level of the articulations of the costal cartilages of the fifth rib.[19] The reason for this specific location is not known.

The etiology of the xiphoid foramen is not well understood, but in standard anatomy textbooks[13,30] it is described as a common variant (Fig. 2).

The incidence of the sternal foramen is not definitely known, since routine sternal roentgenography in living persons has never been done. Greig,[11] however, suggests an incidence of 1–2 per cent in dried skeletal specimens. This anomaly apparently is more common in certain species of apes and monkeys.[19]

SUPRATROCHLEAR FORAMEN

The articular surface of the distal humerus includes a laterally placed eminence, the *capitulum*, and a medial portion, named the *trochlea*. The former structure articulates with the head of the radius, while the latter surface fits within the semilunar notch of the ulna. Just above the trochlea are two depressions or fossae: one anteriorly, called the coronoid fossa, and the second posteriorly, named the olecranon fossa. These fossae function together with the radius and ulna to aid in flexion and extension of the elbow.

The two fossae are normally separated by a bony septum; rarely, only fibrous tissue is present, the bone itself being absent. The resulting structure is then called the "supratrochlear" or "intercondyloid" foramen[31] (Fig. 3, *A* and *B*). This finding is said to occur in 4 per cent of the white race and 13 per cent of the Negro race.[30] It is more common in prehistoric skeletons, apes, and

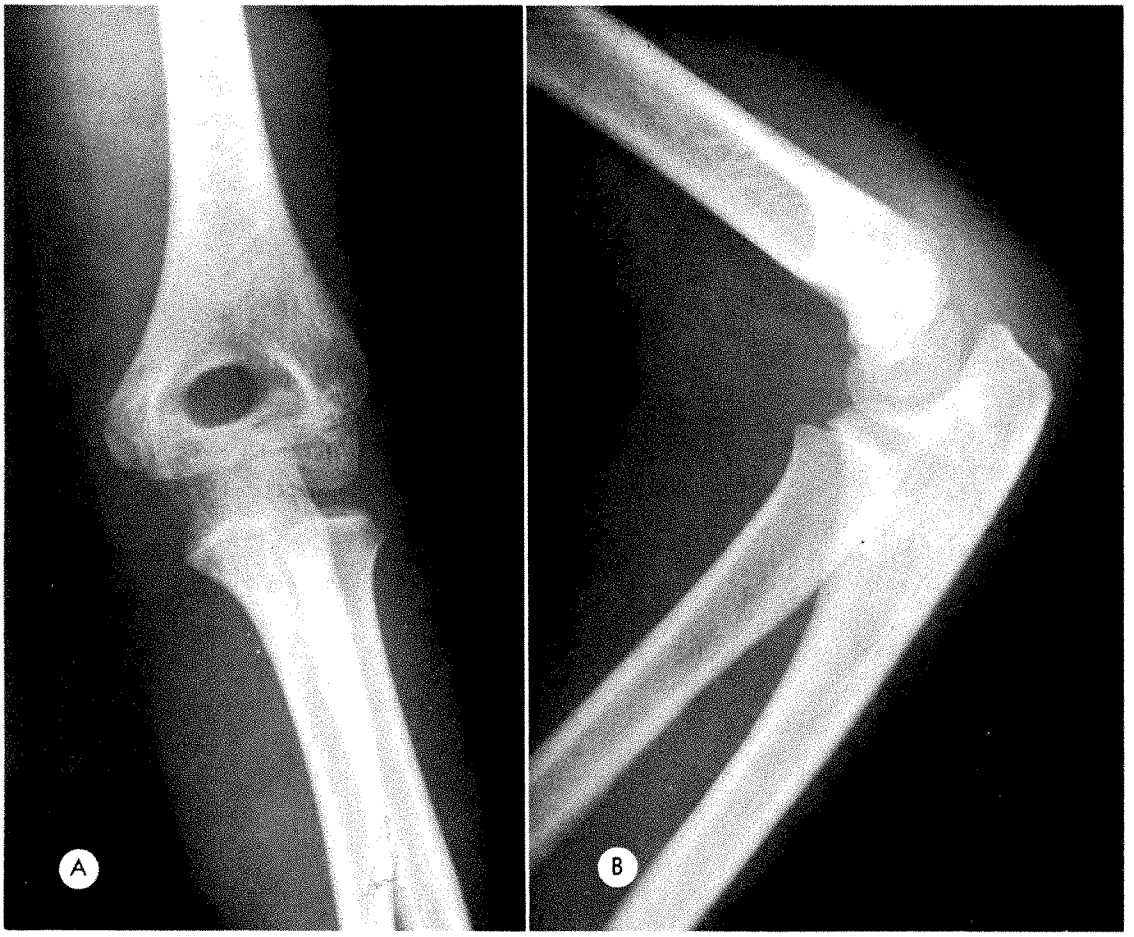


Fig. 3. (*A* and *B*) *Supratrochlear Foramen.* Note that this structure is visualized only in the anteroposterior view of the elbow; the lateral view is entirely unremarkable.

primitive mammals.[4,32] There is a hereditary factor.[16]

INIO-ENDINEAL FORAMEN

The inio-endineal canal or foramen (Fig. 4) is a small channel in the mid-sagittal plane of the occiput at the level of the external occipital protuberance. It is traversed by an emissary vein which connects the external occipital veins with the confluence of venous sinuses.[4] It is said to be present in 44 per cent of skulls[20] and must be differentiated from an osseous opening due to a dermal sinus.

FORAMEN OF THE MIDDLE SUPRACLAVICULAR NERVE

Although Pendergrass and Hodes[22] state that the foramen of the middle supraclavicular nerve is impossible to visualize because of its diagonal direction, Köhler and Zimmer[16] believe that the structure can be seen roentgenographically in 2 to 6 per cent of the population. This foramen or canal occurs in the upper edge of the mid-portion of the clavicle (Fig. 5) and can be seen in 6.6 per cent of dried skeletal specimens.[30] The entity was first described in the German literature by Skarby.[35]

ARCUATE FORAMEN

After emerging from the transverse foramen, the vertebral artery crosses the posterior arch of the atlas. On the floor of the arch a groove is formed, called the vertebral artery sulcus. The artery is covered by the oblique altanto-occipital ligaments. Rarely,

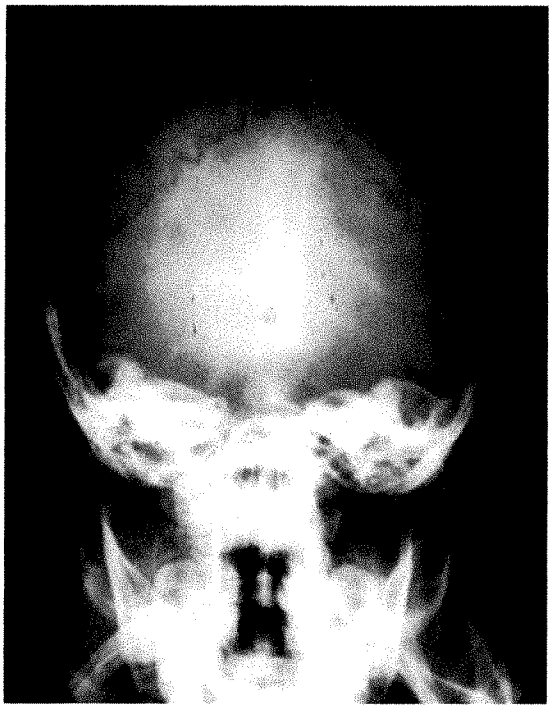


FIG. 4. *Inio-Endineal Foramen.* Towne's view of the skull shows this canal to be located in the mid-portion of the occiput.

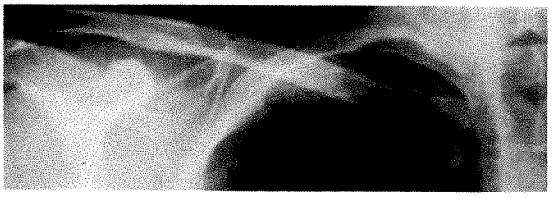


FIG. 5. *Foramen of the Middle Supraclavicular Nerve.* This was, of course, an incidental finding on a routine chest roentgenogram.

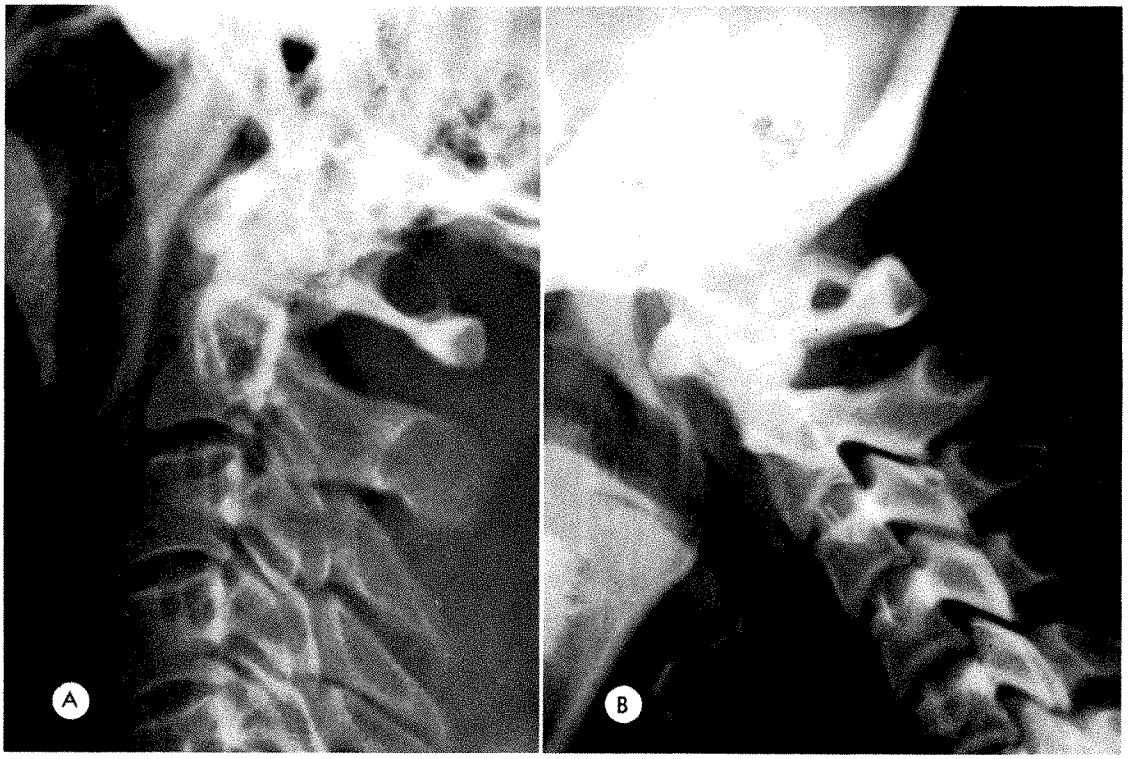


FIG. 6. (*A* and *B*) *Arcuate Foramen.* Note the two different appearances of the ponticulus posticus, one being thin and the other quite thick. The arcuate foramina represent the resulting passage-ways from these bony bridges.

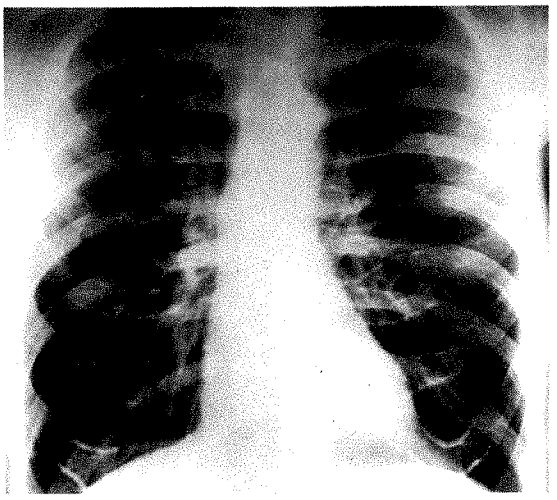


Fig. 7. *Foramen of Bifid Rib.* Note the foramen formed at the junction of the bifid left fifth rib and its adjacent bifid calcified costal cartilage.

an anomalous ossification center occurs in this ligament; subsequent ossification forms a bony arch, called the ponticulus posticus. The resulting foramen is named the arcuate or posterior atlantoid foramen, and contains the suboccipital nerve as well as the vertebral artery.[1,14] The entire complex has been called Kimmerle's anomaly, and occurs in 12.6 per cent of all normal skulls.[6]

There is wide variation in shape, size, and contour of the foramen and its roof; Figure 6, *A* and *B* shows two variations. Pyo and Lowman[27] have emphasized that size of the vertebral artery is in no way correlated with the size of the arcuate foramen.

FORAMEN OF BIFID RIB

Bifurcation of a rib is a congenital anomaly occurring in from .01–.5 per cent of the population.[7,37] When ribs are bifurcated at their sternal ends, they articulate with similarly bifid costal cartilages.[30] Figure 7 shows an example where calcification of a bifid costal cartilage has joined with a bifid rib to create a foramen. It is probable that the foramen is functionless, and is undoubtedly filled with fibrous tissue. It is generally believed that the cause

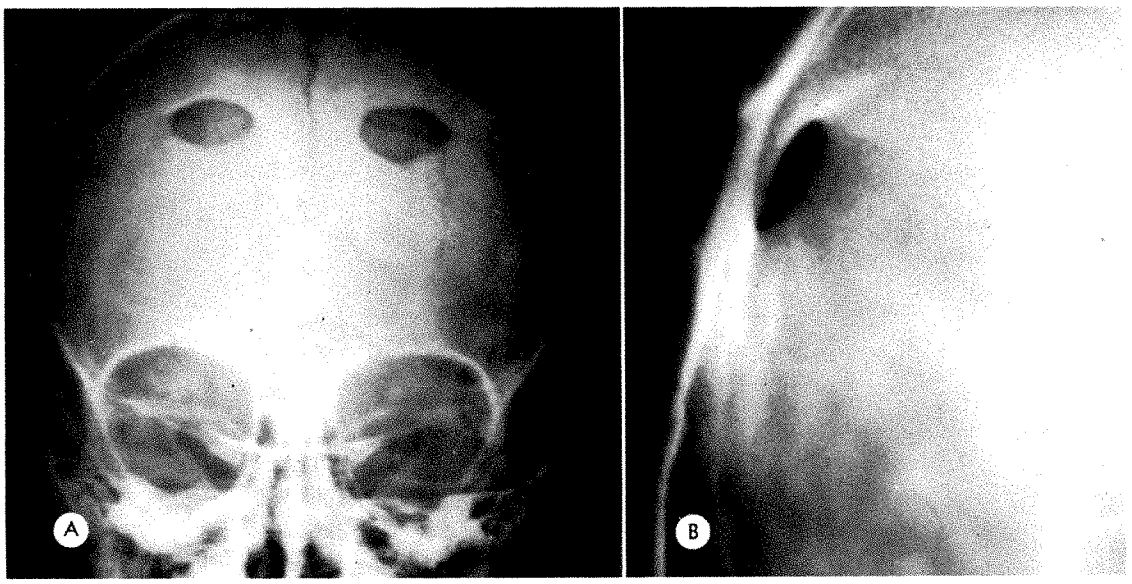


Fig. 8. (*A* and *B*) *Parietal Foramina.* Note the characteristic location and appearance in both anteroposterior and lateral views of these enlarged foramina.

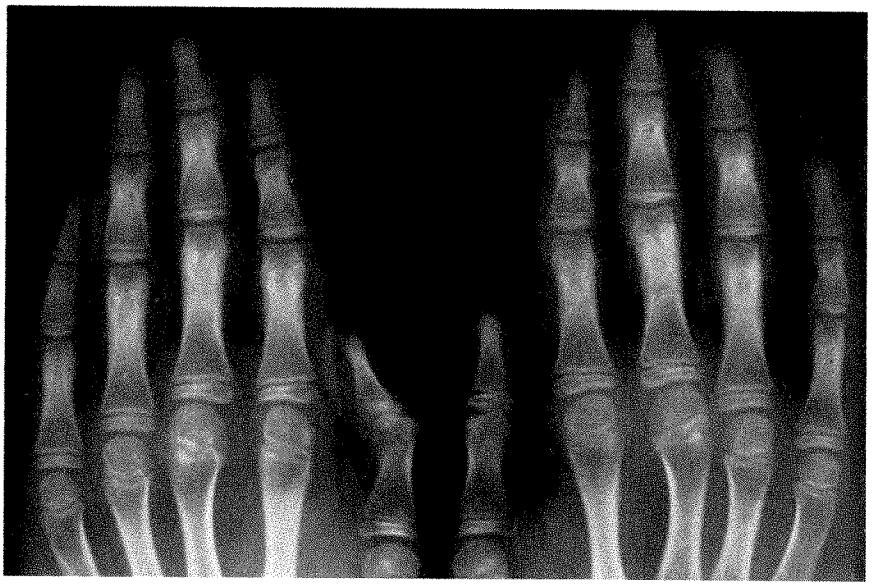


FIG. 9. *Vascular Foramina of the Carpal Phalanges.* These canals can be seen in the waist-like constricted areas of the middle phalanges of the second through fifth digits bilaterally.

of costal cartilage calcification is unknown; this calcification may take place any time in adult life and is unrelated to disease.[15]

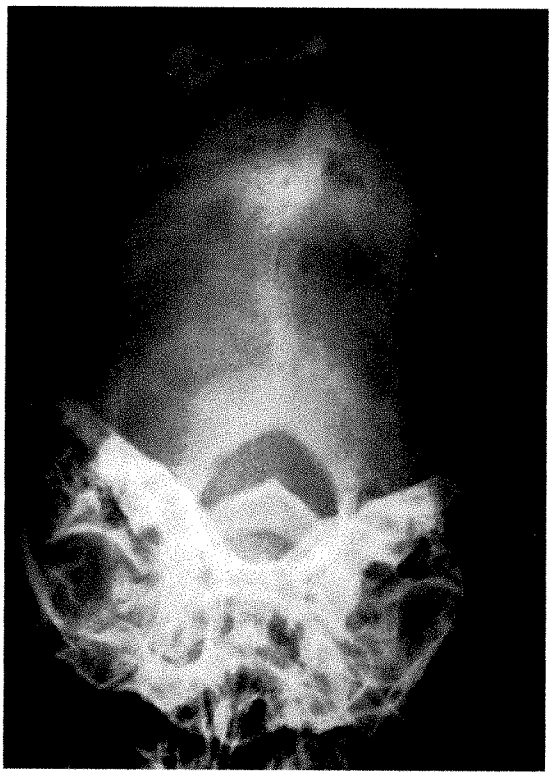


FIG. 10. *Posterior Condylar Fossa.* This finding, more marked on the patient's left, is usually best seen in the occipital view of the skull.

PARIETAL FORAMINA

This entity is one of the best known and most thoroughly studied of all bone anomalies. Since the subject has been covered well in both textbooks[24] and medical journals,[12,23,25,38] only a brief review is made here.

Parietal foramina are defects in the parietal bones, located 2 fingers ventral to the apex of the lambdoid suture and 1 cm. to the left and right of the sagittal suture.[16] They occur in 60 per cent of people, but in 90 per cent of these the defects are less than 1 mm. in diameter.[21] Rarely, however, the defects may measure up to 5 cm. in diameter. The foramina transmit emissary veins connecting the occipital veins with the superior sagittal sinus.[33] There is a definite hereditary tendency; Goldsmith[9] found parietal foramina in innumerable members

of the Catlin family, and termed the phenomenon the "Catlin mark."

The finding is of no real clinical significance, but may present a rather bizarre appearance on the skull roentgenogram (Fig. 8, *A* and *B*).

VASCULAR FORAMINA OF THE CARPAL PHALANGES

Although nutrient foramina occur in all parts of the skeleton, they are not often visualized and perhaps even less frequently recognized. In Figure 9 can be seen the characteristic punched-out holes at the levels of the waist-like constrictions of the middle phalanges of the fingers; this appearance is typical of nutrient canals.[16] According to Caffey,[4] these foramina are rarely seen in the distal or proximal phalanges.

2. PSEUDOFORAMINA

A foramen is defined as "a natural hole or passage, especially one into or through a bone."[5] Foramina, then, are seen both on roentgenologic examination and on inspection of the anatomic bone specimen itself. The following conditions are termed "pseudoforamina," for although they simulate a hole roentgenographically, inspection of the actual bone specimen reveals no such osseous defect.

POSTERIOR CONDYLAR FOSSA

On either side of the foramen magnum behind the occipital condyles lie the posterior condylar fossae. These depressions in the base of the skull articulate with the superior posterior part of the lateral masses of the atlas when the head is bent backward. The roentgen anatomy and clinical significance of this entire region was described by Haas[13] in 1957. According to his report, the appearance of the fossae is variable; they may be absent, shallow, small, large, symmetric or asymmetric.

The translucent shadows of the fossae do not correspond to actual foramina; they are due to the extreme thinness of the bone in this area. Occasionally, the fossae may contain a tiny vascular canal. These structures are usually best demonstrated in the occipital view of the skull (Fig. 10).

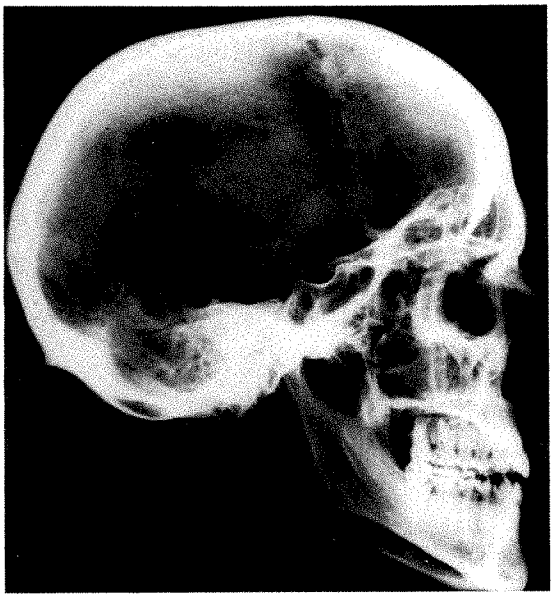


FIG. 11. *Squamosal Portion of the Temporal Bone.* Lateral view of a dried skull specimen shows the extreme thinness of the squamosal portion of the temporal bone, simulating a large bony defect.

SQUAMOSAL PORTION OF TEMPORAL BONE

The squamosal portion of the temporal bone is flat, thin, often translucent,[30] and is the thinnest bony part of the lateral wall of the cranial vault.[16] Often a lateral view of the skull suggests the complete absence of bony substance in this area (Fig. 11). Especially when physiologic sclerosis accompanies the squamosal suture, the squamosal bone itself may simulate a large foramen.

ILIAC FOSSA

Usually no cancellous bone is found in iliac fossa portions of the pelvic bones; only a thin sheet of compact bone is present, making the structure semitransparent.[30] As a result, roentgenograms of the pelvis may suggest a large defect of bone in the middle of the ilium; in particular, an oblique view may emphasize this "pseudoforamen" (Fig. 12).



FIG. 12. *Pseudoforamen of the Iliac Fossa.* Oblique view of the pelvis shows a large bone defect simulated by the normal thinness of the iliac fossa.

BODY OF THE SCAPULA

Above and below the spine of the acromion are thin portions of the scapula known as the supraspinatus and infraspinatus fossae. These areas are unusually thin and may appear translucent.[8] Occasionally, there is an actual absence of bone, and the adjacent muscles are separated only by fibrous tissue.[10] Again, varying the position of the anatomic part emphasizes this normal "hole-like" appearance (Fig. 13).

LATERAL MALLEOLAR FOSSA

The distal fibula terminates with the lateral or fibular malleolus. On the medial aspect of this malleolus, in the location of the articulation with the talus, there is a small excavated area known as the malleolar fossa.[8,30] Usually, this depression is quite inconspicuous, but occasionally it may simulate a hole or lesion within the bone (Fig. 14, *A* and *B*).

HEAD OF THE HUMERUS

Brailsford[2] states that in the head of the humerus there are areas of radiotransparency of various sizes—sometimes with well defined margins. These are seen unassociated with bone disease and are of themselves of no clinical significance. It has been observed that when the arm is held in abduction, the greater tuberosity area resembles a hole or foramen (Fig. 15).

PSEUDOCYSTIC AREA OF OS CALCIS

Sirry[34] in 1951 described a triangular shaped area of diminished bone architecture in the anterior portion of the os calcis which he termed the "pseudocystic triangle." Caffey[4] states that the radiolucency may be either triangular or circular, and that it is seen in 10 per cent of children over 7 years of age. Of 1,200 cases, Sirry found pronounced evidence of this phenomenon in 7 per cent, moderate changes in 21 per cent and very faint changes in 71 per cent. The anatomic specimens show deficiency of osseous trabeculae,[16] but no specific disease is seen. An example of this finding is shown in Figure 16.

RADIAL TUBEROSITY

The tuberosity of the radius is a cortical roughening present on the anteromedial aspect of the proximal portion of the bone; it receives the insertion of the biceps ten-

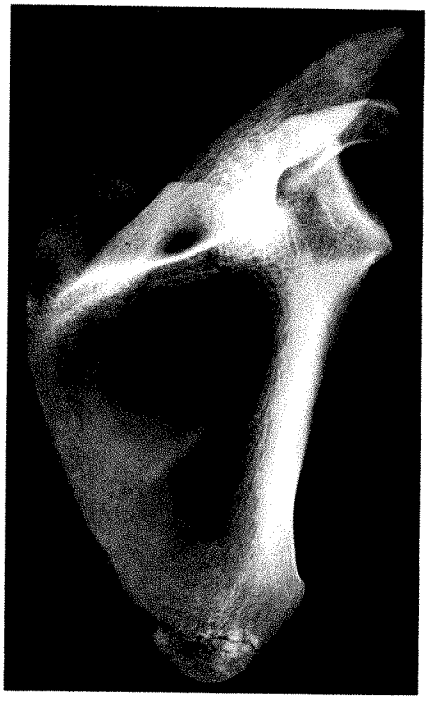


FIG. 13. *Pseudoforamina of the Scapula.* Anteroposterior roentgenogram of a dried scapular specimen showing the marked thinning of the supra- and infraspinatus fossae. Often these areas simulate large foramina.

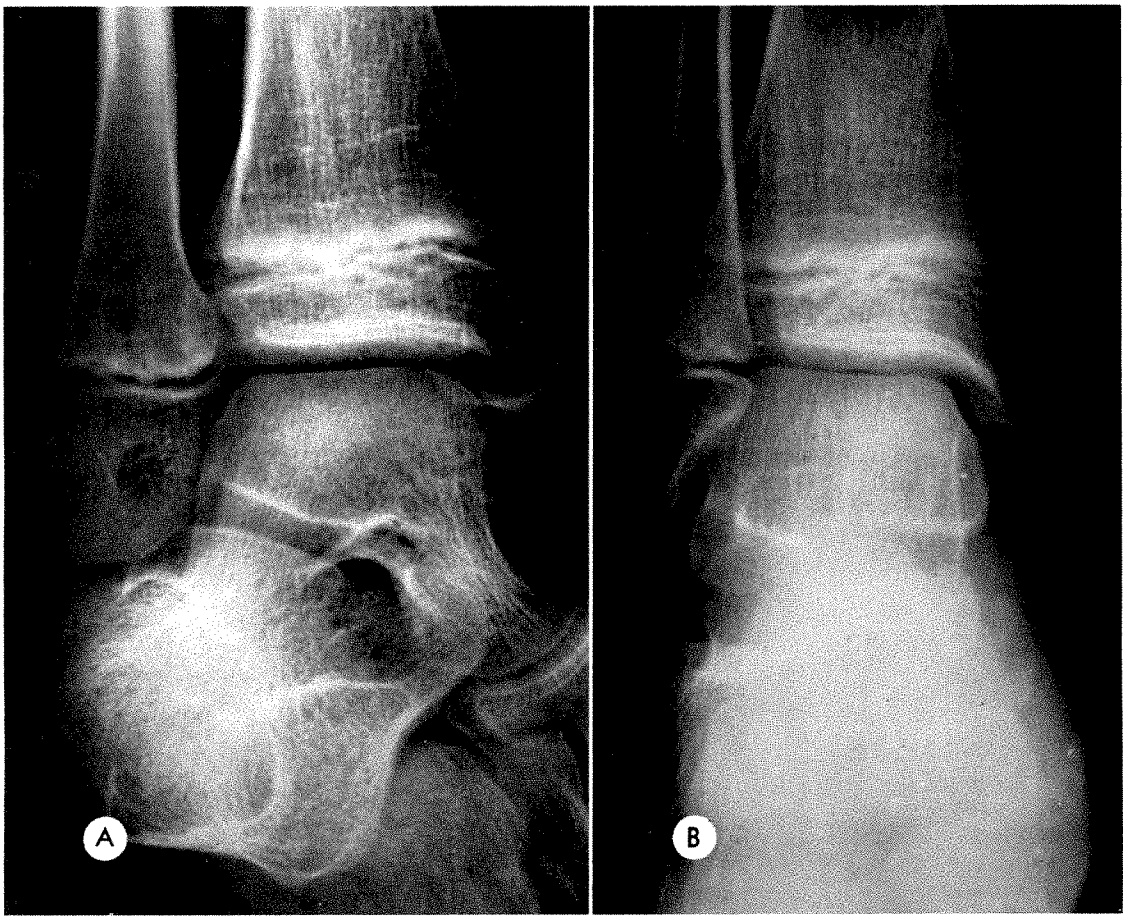


Fig. 14. (*A* and *B*) *Lateral Malleolar Fossa.* Anteroposterior and lateral views of the ankle show the lateral malleolar fossa simulating a foramen when the structure is seen *en face.*

don. Köhler and Zimmer[16] state that there is sometimes seen at this site a defect, depression or pit. This is an unusual normal variation; microscopic sectioning of the bone itself reveals no abnormalities. This finding appears to be more commonly seen in childhood (Fig. 17, *A* and *B*).

Nutrient Pseudoforamen of the Distal Femoral Epiphysis (Fovea Cribrosa Vasorum Intercondyloidae Femoris)

In 78 per cent of human femora, a distinct fovea of from 3 to 8 mm. in diameter

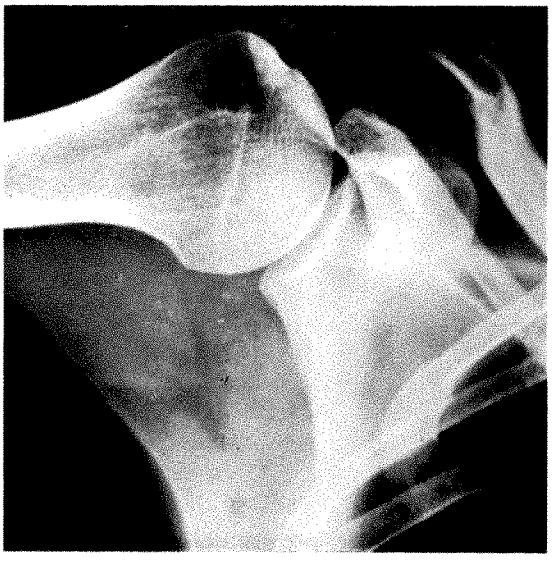
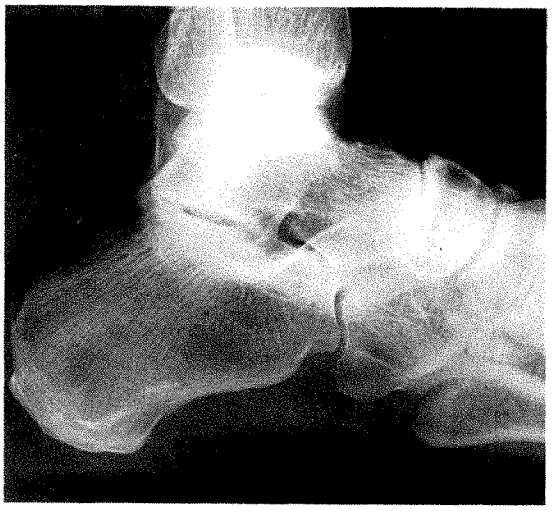


Fig. 15. *Pseudoforamen of the Greater Tuberosity of the Humeral Head.* This is especially prominent when the arm is held in abduction.



is centrally located in the anterior inferior portion of the intercondylar fossa between the attachments of the cruciate ligaments. Within this fovea are contained 5 to 15 nutrient foramina, through which pass middle genicular branches of the popliteal artery. In a rather extensive study of this region, Rogers and Gladstone[28] found that in 78 per cent of the cases, the fovea is shallow and inconspicuous. In the remain-

FIG. 16. *Pseudocystic Area of Os Calcis.* Note the area of diminished bone density in the anterior portion of the os calcis. This is not abnormal.

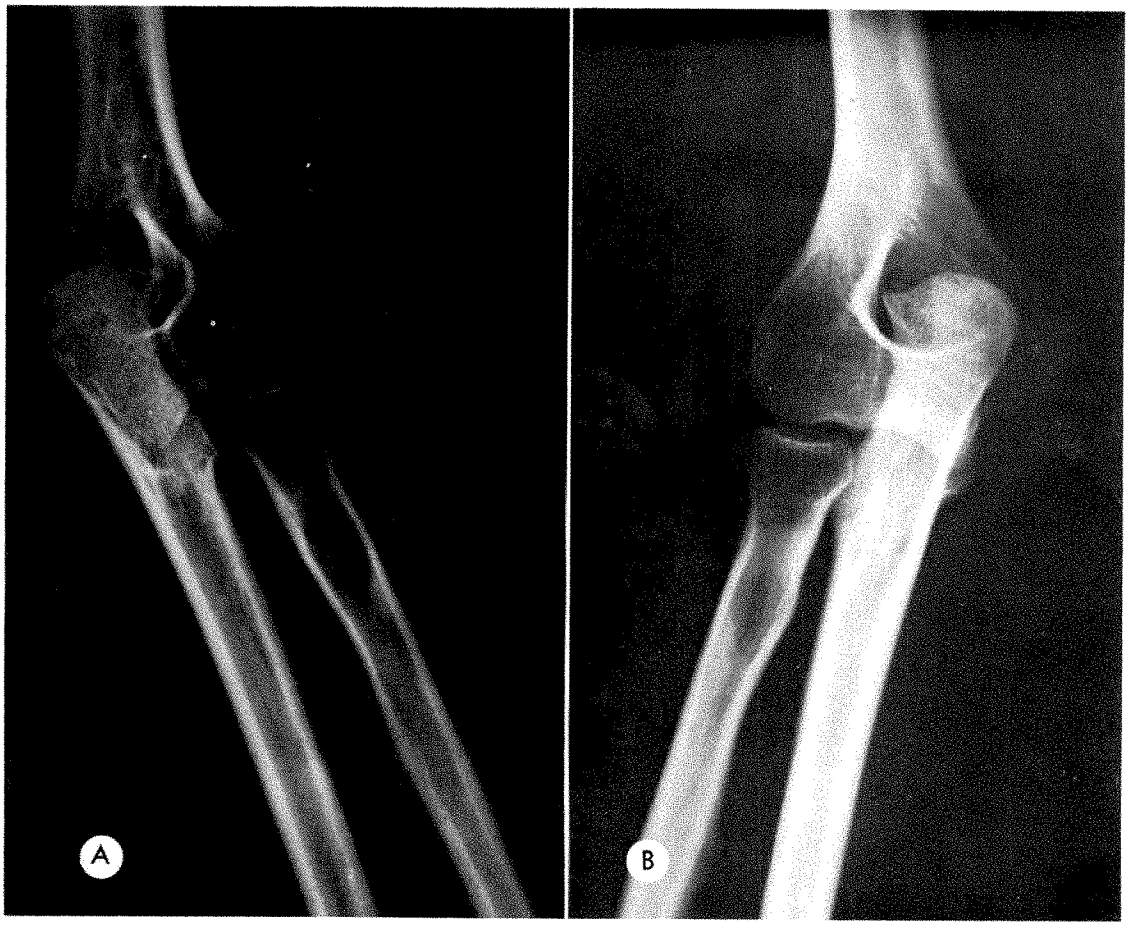


FIG. 17. (*A* and *B*) *Radial Tuberosity.* Oblique views of the forearm reveal how a depressed radial tuberosity can simulate a foramen or bone lesion.



Fig. 18. *Nutrient Pseudoforamen of the Distal Femoral Epiphysis.* Note the symmetric bilateral rarefactions in the intercondylar region of the distal femoral epiphysis.

ing 22 per cent, however, the fovea is deep, measuring 3 to 5 mm. in depth, and causes an area of rarefaction on the roentgenogram (Fig. 18). It is this rarefaction that Caffey[4] terms the "nutrient foramen of the distal femoral epiphysis." This structure is called a "pseudoforamen" here, however, since it is actually a bony depression rather than a true foramen. Rogers and Gladstone coined the more exact term of "fovea cribrosa vasorum intercondyloidae femoris."

PSEUDOFORAMEN OF THE SUPERIOR LATERAL TIP OF THE SCAPULA

Figure 19 shows an apparent bone defect in the superior lateral aspect of the scapula. Köhler and Zimmer[16] warn that this represents a normal thinning of bone, and is not to be confused with a pathologic lesion. The radiolucent spot is bounded by the glenoid fossa, the coracoid process, and the lower border of the scapular spine.

3. DEVELOPMENTAL DEFECTS

While incomplete ossification within the central portion of a bone forms a foramen, a similar abnormal ossification process occurring at the periphery of a bone can be called a "developmental defect." Three such examples are presented. One is well known to many radiologists; the others have never been described in the American literature.

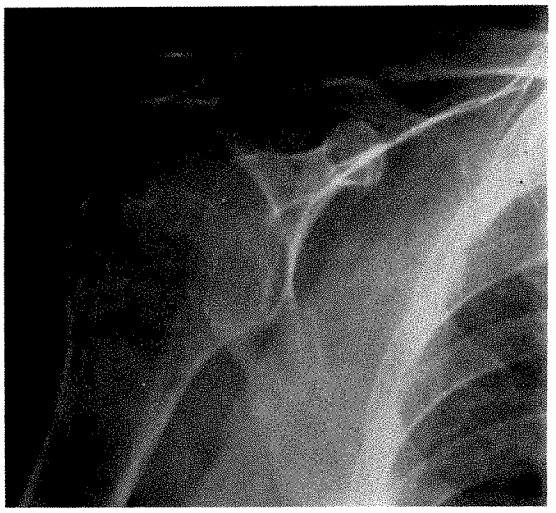


Fig. 19. *Pseudoforamen of the Superior Lateral Tip of the Scapula.* Note the tiny radiolucent spot surrounded by the glenoid fossa, the coracoid process, and the scapular spine.

RHOMBOID FOSSA OR NOTCH

In 1937 Pendergrass and Hodes[22] first described the rhomboid fossa, a rough, irregular depression of the medial end of the clavicle. This area lies 1 to 1.5 cm. from the sternoclavicular junction on the inferior surface of the clavicle, and serves as the attachment for the costoclavicular (rhomboid) ligament (Fig. 20). The finding occurs in 10 per cent of the population, and may resemble a destructive bone lesion.

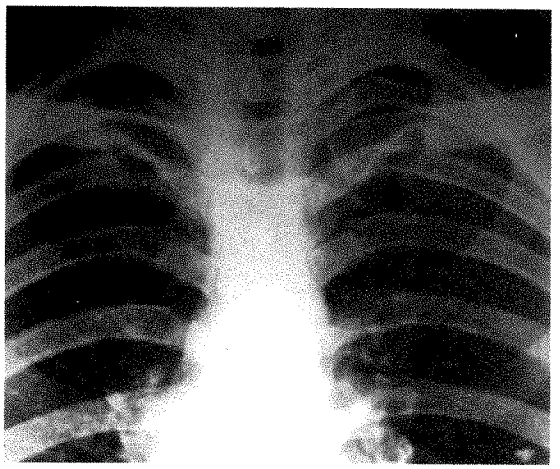


Fig. 20. *Rhomboid Fossa.* Note the bilateral rhomboid notches at the medial aspects of the undersurfaces of the clavicles.

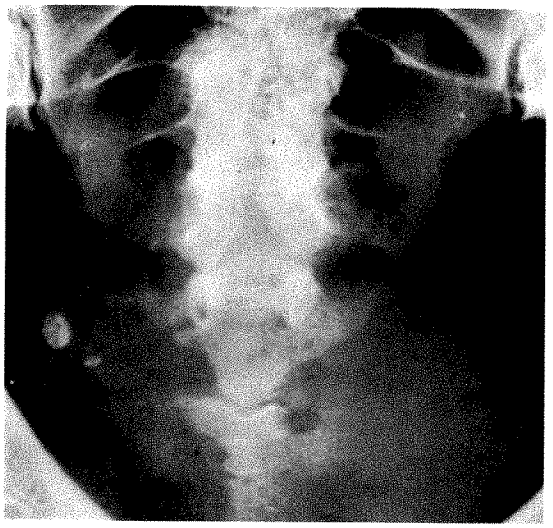


FIG. 21. *Variation of the Sacral Coccygeal Area.* There is bilateral incomplete union of the transverse processes of the first coccygeal segment and the inferior sacral angles; therefore, the fifth sacral foramina are only partially formed. It is obvious how complete lack of development of this bone ridge could simulate a large osseous defect.

INFERIOR SACRAL NOTCH

The adult sacrum is a large solitary triangular shaped bone articulating with the lumbar spine superiorly, the hip bones laterally, and the coccyx inferiorly. It is the result of complete fusion of 5 original sacral vertebrae. Normally, the sacrum encompases 4 pairs of anterior sacral foramina and 4 pairs of posterior foramina; laterally the lower end of the sacrum terminates in projections, the inferior lateral angles. Medial to each angle is an indentation of bone, the inferior sacral notch. Usually this notch is converted into a foramen by union of the inferior sacral angle and the transverse process of the first coccygeal segment; passing through this foramen is the anterior division of the fifth sacral nerve.[6,10,30]

In perhaps 50 per cent of the population, however, anomalies in the sacrococcygeal area exist.[18] The first coccygeal segment may fuse completely with the sacrum; the union of the transverse process of the first coccygeal segment and the inferior sacral angle may be incomplete (Fig. 21); or the direction of the coccyx in relation to the sacrum may be deviated in almost any manner—anteriorly, posteriorly, or obliquely.[16,32]

If one transverse process of the first coccygeal segment fails to fuse with the sacral angle while the opposite side does

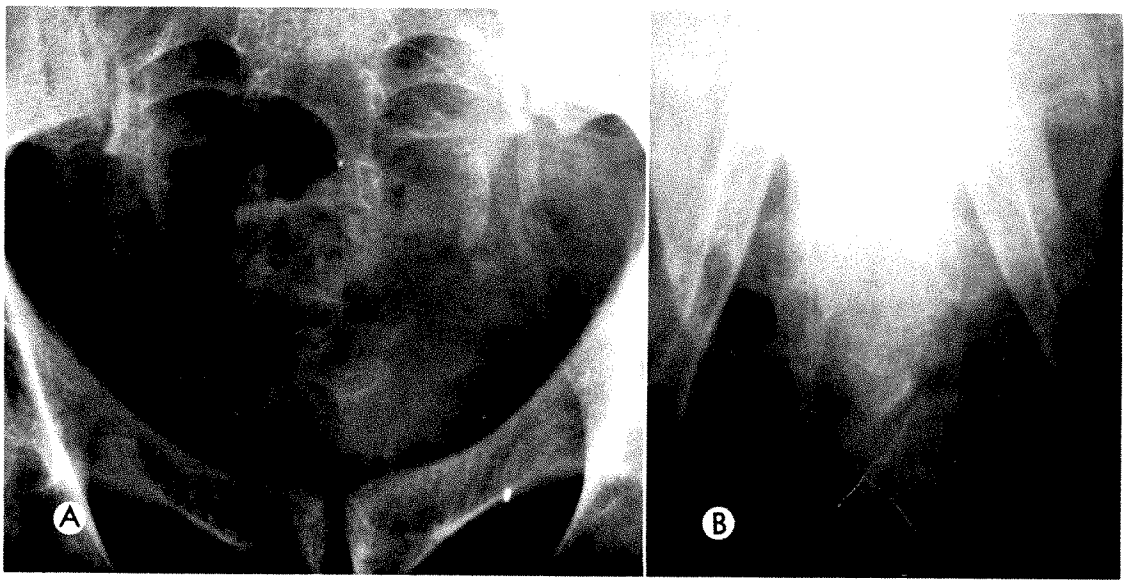


FIG. 22. (*A* and *B*) *Prominent Inferior Sacral Notch.* This patient was originally thought to have a sacral tumor on the left. Note, however, the angulation of the coccyx to the left, and the marked prominence of the sacral notch. These findings are variations of normal.

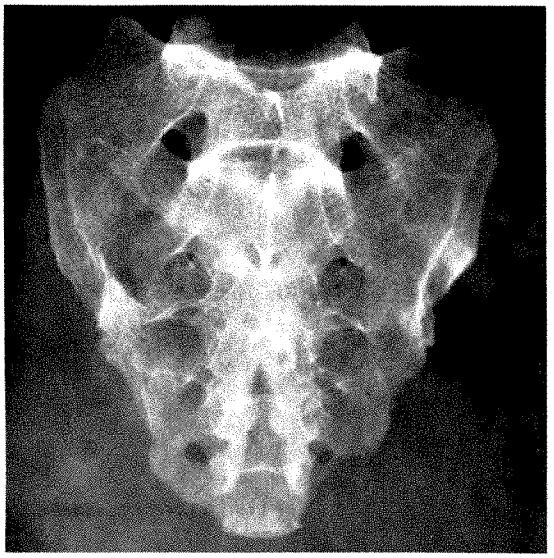


Fig. 23. *Prominent Inferior Sacral Notch.* Note the similarity in appearance between this roentgenogram of dried skeletal specimen and the case illustrated in Figure 22, *A* and *B*.

and if the coccyx is specifically angled to the unfused side, a normal bony defect representing the open inferior sacral notch may appear (Fig. 22, *A* and *B;* and 23). This notch may be so prominent and so asymmetric that it may well simulate a tumor of the sacrum. Variations of this type of appearance have been described in several reports,[36,39] but the possible clinical implications have never been described.

PARAGLENOIDAL SULCUS

Lateral to the sacroiliac joint of the pelvis may exist a defect in the bony contour called the paraglenoidal sulcus[16] (Fig. 24). This groove surrounds the superior branch of the gluteal artery and provides for the insertion of deeper strands of the sacroiliac ligament. The incidence of this minor defect in bone ossification is unknown. It may simulate a destructive lesion of bone.

DISCUSSION

"Variability," stated Ryle,[29] "is one of the most distinctive and necessary attributes of life." It behooves the roentgenologist to recognize these variabilities since his awareness of them may prevent a patient from receiving treatment for a normal finding which simulates a lesion.

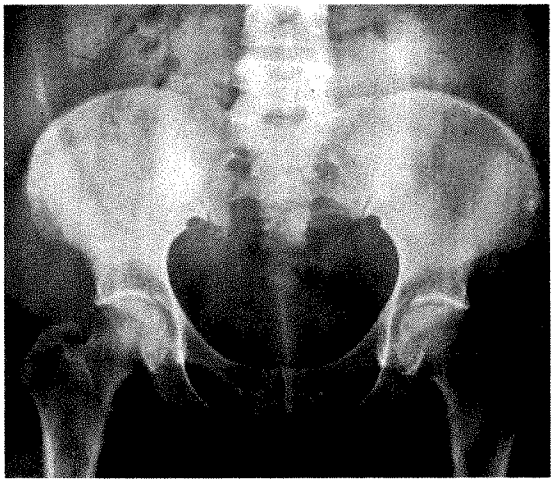


Fig. 24. *Paraglenoidal Sulcus.* Depressions in the cortical margins of the roof of the true pelvis are seen in the regions lateral to the sacroiliac joints.

Some of the foramina and pseudoforamina discussed may be well known to some. However, several of the entities, including the sternal foramen and the inferior sacral notch, to the best of the author's knowledge, have never before been mentioned in the literature. The particular case in Figure 22, *A* and *B* was originally diagnosed as a sacral tumor before the actual anatomy and normal variations of the sacral region were fully realized.

It should be emphasized that no attempt has been made to review all bony foramina and pseudoforamina. Caffey[4] lists numerous nutrient vessels occurring in many different bones, and anatomy texts will reveal many others. The radiologist should always be cognizant of the occurrence of these entities when he is confronted with a radiolucency of bone.

SUMMARY

Various foramina, pseudoforamina, and developmental defects of bone are discussed and illustrated. Several, including the sternal foramen and the inferior sacral notch, are reported for the first time in the

medical literature. These findings are variations of normal; their clinical importance lies in their differentiation from osteolytic bone lesions.

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LYMPHANGIECTASIS*

BENIGN AND MALIGNANT

By ARTHUR S. TUCKER, M.D.
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LARGE, endothelium-lined spaces and channels have been found at autopsy in patients dying of various diseases with different clinical manifestations. The pathologist is apt to designate this condition as lymphangiectasis or hemangiectasis, depending upon whether the spaces grossly contain lymph fluid or blood. The radiologist cannot differentiate on his roentgenograms between lymph and blood collections but from the gross distribution of the lesions he is in a position to advise regarding the extent and prognosis of the disorder.

In some instances the profuseness of the endothelial tissue on histologic examination leads the pathologist to a diagnosis of lymphangiomatosis or hemangiomatosis; however, evidence that the tissues are the result of tumor growth is scanty. The proliferation of endothelial cells seems rather to be a response to mechanical stress, and the architecture is similar to that which results from tying off a lymph duct. While there are varying descriptions of the microscopic picture from different authors, none has found evidence of rapid proliferation of the tissues or of metastatic tendencies.

In the presence of such varied anatomic involvement, the radiologist must be prepared to see abnormalities ranging from minute or insidious to gross or overwhelming. In general, the roentgenographic findings in published accounts fall into three categories: pulmonary, osseous, and diffuse or multiple-system. The three types are related by their histologic similarity; they may be entirely separate entities from an etiologic standpoint. Certainly their clinical courses differ markedly.

To call attention to the three types of the disease process, a brief description and an illustrative case of each are presented.

PULMONARY LYMPHANGIECTASIS

Congenital cystic disease of the lungs usually is related to abnormal development of the bronchi. A number of cases have been described, however, where the histologic structure indicated an anomaly of lymphatic origin. Frank and Piper[6] in 1959 collected 21 such cases in the literature dating back to Virchow[21] in 1856, and added 2 others. Carter and Vaughn,[4] more recently, have reported 1 more. It is indicated in the published reports that the congenital defect is found only in fetuses and newborn infants. Carter and Vaughn conclude that "the roentgenographic findings in congenital lymphangiectasis of fine granular densities distributed throughout the lungs simulate those of hyaline membrane disease and fetal atelectasis." Frank and Piper state "It is apparent from a review of all cases that this entity, no matter by what name it is called, must be included with other disease processes responsible for neonatal cyanosis and, unfortunately, can be positively identified only by means of autopsy."

We have had a similar experience with cystic lymphatic disease in the perinatal period; however, we should like to suggest that the pulmonary condition may be neither so confined as to age incidence, nor necessarily so fatal, because of our experience with an older patient.

Laurence[18] has pointed out that in over half of the autopsies performed on cases of congenital pulmonary lymphangiectasis there are encountered other major congenital abnormalities as well. Giammalvo's[7] case showed, in addition to congenital pulmonary cystic disease of lymphatic origin with extensive atelectasis and developmental retardation of the lung, a

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congenital heart lesion characterized by atresia of the aortic valve, hypoplasia of the ascending aorta, a diminutive left ventricle, sclerosis of the mitral valve with mitral stenosis, dilatation and hypertrophy of the left auricle, and hypertrophy of the right ventricle.

Our patient also manifested a congenital heart lesion, but of an entirely different nature.

ILLUSTRATIVE CASE

Case 1. A boy of $3\frac{1}{2}$ years of age who had been known to have a congenital heart defect since birth was studied at Babies and Children's Hospital because of periods of cyanosis and an episode of loss of consciousness.

He had weighed 6 lb. at birth, which occurred after a 36 week gestation; otherwise, the antenatal history was normal. From birth he had been observed to have a large head, but his mental development had been considered to be normal. During his first year of life he was noted to be bluish, particularly in a circumoral distribution, but since then cyanosis occurred only after he exerted himself.

The child was well developed and well nourished. His height measured at the fifteenth percentile, his weight in the sixtieth. The precordium was prominent, particularly on the left. A systolic thrill was felt to the left of the sternum in the third intercostal space, where there also was a Grade 3 harsh systolic murmur. P_2 was present but soft.

The electrocardiogram showed a marked right axis deviation and a sinus arrhythmia. At

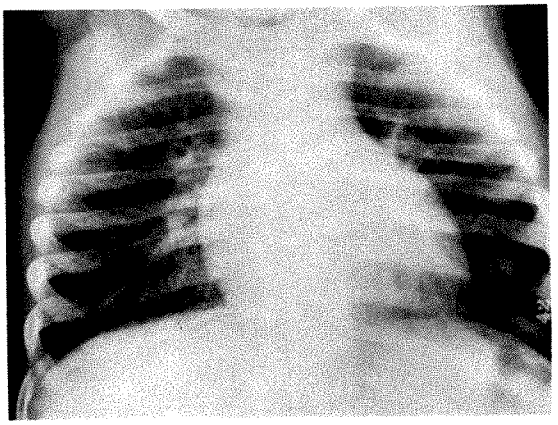


Fig. 1. Case 1. Preoperative roentgenogram of chest at the age of 3 years, 11 months.

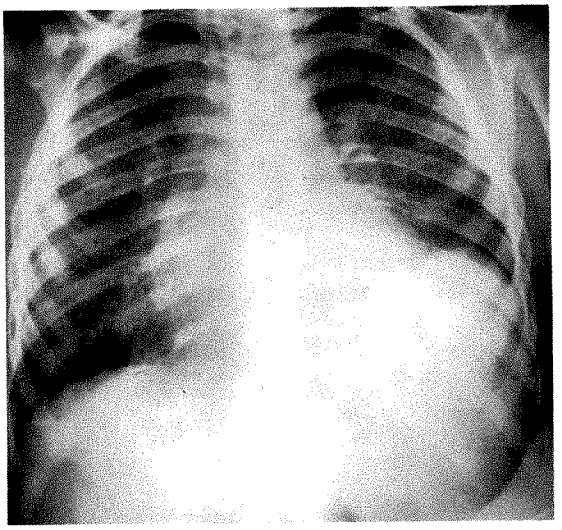


Fig. 2. Case 1. Chest roentgenogram with the mobile unit 4 hours postoperatively. Lung markings are more pronounced than in Figure 1. Elevation of the base of the left lung was due to a pleural collection of blood.

heart catheterization very high pressures were encountered in the right ventricle, ranging from 100 to 130 mm. of mercury, systolic. Pulmonary artery pressures were normal, 18 to 25 mm. systolic.

Although roentgenograms showed increased rather than diminished pulmonary markings (Fig. 1), the other findings pointed to marked pulmonary stenosis. An open-heart operation was carried out just before the boy reached 4 years of age and confirmed the diagnosis. The pulmonary valvular ring was very small, and the pulmonary artery distal to the valve was also of small caliber. The valve itself showed fusion of all three commissures so that the opening was a mere hole in a diaphragm. Valvulotomy was carried out and as a result the pressure in the right ventricle dropped from 120 to 60 mm. of mercury.

Postoperatively, the pulse rate rose until it reached 180. Four hours after the completion of the operation the patient developed acute respiratory distress. Bag-and-mask breathing was instituted, but the patient's heart stopped. A roentgenogram made with the mobile unit showed a fluid accumulation of blood at the base of the left lung (Fig. 2). The surgeon reopened the chest and by cardiac massage, defibrillation, and multiple medications over a period of 1 hour and 15 minutes restored the heart beat. The respiratory and central nervous

systems continued to function poorly, however, and 11 hours after the operation the patient was pronounced dead.

At autopsy no disruption was found at the operative sites. Small hemothoraces were present, 50 cc. on the left, 100 cc. on the right. The heart was large, weighing 150 gm., with right ventricular hypertrophy. The pulmonic ring measured 4.0 cm. in circumference, the valve margins 3.0 cm. The lungs were unusually firm and heavy and showed increased amounts of fibrous tissue around the pulmonary vessels and bronchi. Microscopically, there was an increase in the interlobular and peribronchial connective tissue, and there were numerous irregular spaces resembling lymphatic channels within the connective tissue (Fig. 3). The pathologic diagnosis was interstitial pulmonary lymphangiomatosis and fibrosis in a postoperative patient with pulmonary stenosis.

Comment. The pulmonary lesion appeared histologically indistinguishable from that found at autopsy in the perinatal respiratory distress patients who were found to have cystic endothelium-lined spaces. How much this defect contributed to the patient's symptomatology is debatable in view of the severe pulmonary arterial constriction which was found at surgery. Nevertheless, the boy's physical activity and growth had not been notably limited.

OSSEOUS LYMPHANGIECTASIS

Involvement of the skeleton by tumors of endothelial origin sometimes leads to widespread or bizarre degrees of osteolysis. Inasmuch as the histologic nature of the tissues is similar whether the involved channels belong to the lymphatic or the vascular system, there may be differences of opinion as to whether the lesions should be classified as lymphangiomas[12,19] or hemangiomas.[8] Some reported cases have not been thoroughly studied, or not adequately explained after dissection, and have consequently been described under such titles as massive osteolysis,[9,11,14,15,20] essential osteolysis,[3] acute spontaneous absorption of bone,[9] phantom bone,[9] vanishing bone,[17] and disappearing bone.[1,10,15] The eponym Gorham's disease[11] has been applied because Gorham was the senior author in the compilation of the largest series

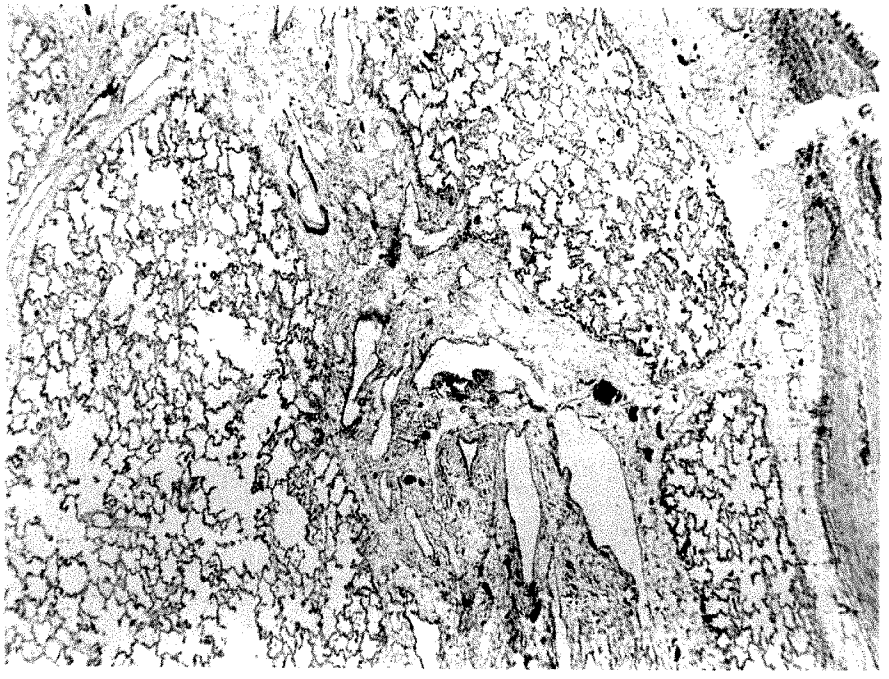


FIG. 3. Case 1. Histologic section of lung from autopsy (×30). Heavy, irregular bands of fibrous tissue are distributed through the otherwise rather normal pulmonary parenchyma. At the bottom of the section are a number of open spaces which are lined with endothelial cells.

from the literature (22 cases) and the personal examiner, with Stout, of tissues from 8 cases.

The nature of the disorder is such as to render it easily diagnosable by roentgenographic examination. The earliest recorded case, nevertheless, antedates the discovery of the roentgen ray by many years. The first diagnosis made in life is attributed to Jackson in two reports.[2,13] His patient was known to live with the affliction for 52 years. Excerpts from the two reports, 34 years apart, provide the following case history.

ILLUSTRATIVE CASE

Case II. "Mr. Brown, a worthy and industrious provision dealer in Derne Street, now thirty-six years of age, in his eighteenth year had the misfortune to have the right humerus fractured nearly in the middle. He was holding, under disadvantages, an enraged cow by the horns; in some unexpected movement of the unruly animal, both fell, and the bone of his right arm was broken in the fall.[2]

"Subsequently, and before he had recovered entirely, he broke his arm, at the same point, a second time, while holding a plough in the field. A short time afterward, he slipped on the floor from snow on his feet, and fractured the bone a third time. The last accident was not more than two months from the occurrence of the first.[13]

"Notwithstanding a most vigilant and untiring devotion to the injured limb, the divided extremities would not adhere; and, to the surprise of the medical attendant, the shaft of each part of the divided bone began to diminish in size, and shorten in length.[2]

"There was never any tendency to a union of the broken bone, but the humerus began slowly and painlessly to disappear, small fragments of it breaking off, without accompanying inflammation of the soft parts, or any fistulous or suppurating sore, or orifice. The skin of the arm was never broken, and no piece of bone or part of the humerus was ever taken away, or came out unassisted. . . . The process of absorption covered a period of twelve years, at the end of which time the limb had attained a condition in which it permanently remained until the day of his death, at the age of 70 years. This occurred at the Boston Lunatic Hospital, on February 11th, 1871, and was caused by double pneumonia. . . .[13]

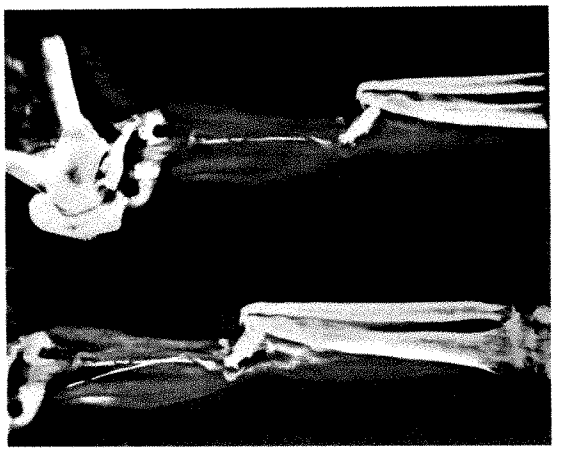


Fig. 4. Case II. Two studies of the "boneless arm" from the Warren Museum of Anatomy. A thin irregular strand of bone is all that remains of the humerus, although the muscles of the arm remain intact. An artefact caused by a scratch crosses the lower reproduction. The proximal ends of the radius and ulna are also noted to have been irregularly absorbed. (Reproduced by courtesy of Dr. J. B. Dealy, Jr.)

"The changes which sometimes take place in the articulating extremities of bones in certain cases of locomotor ataxia are, perhaps, to be remembered in this connection, though nothing suggestive of that, or some similar disease, so far as Mr. B.'s general health was concerned, was ever noticed, beyond a disturbance of the nervous centres, leading to an insanity of the form termed Chronic Circular Mania. . . ." [13]

Comment. This patient was persuaded to offer his arm to be studied after his death, and it was carefully dissected by Prof. Thomas Dwight of Harvard Medical School and preserved in the Warren Museum in Boston. Many years later it was roentgenographed by Dr. M. C. Sosman. The roentgenogram of the specimen is preserved in the teaching file of the Peter Bent Brigham Hospital, and is herewith reproduced by courtesy of Dr. J. B. Dealy, Jr. (Fig. 4).

DIFFUSE LYMPHANGIECTASIS

Involvement of *lungs* by angiomatosis appears to be a fatal process although one of our patients lived without specific symptoms for 4 years and might have survived considerably longer if he had not been operated upon for a severe accompanying

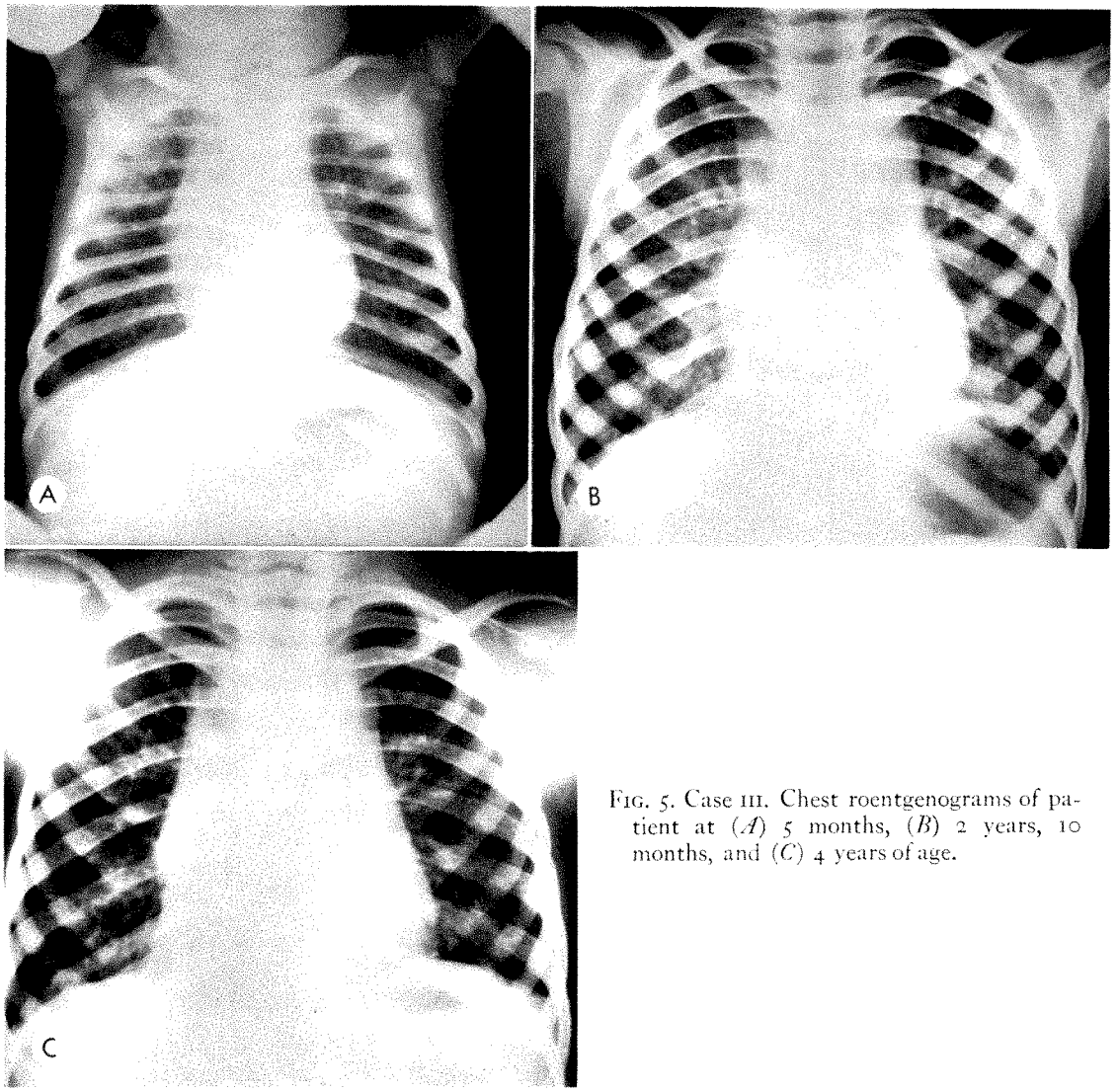


FIG. 5. Case III. Chest roentgenograms of patient at (*A*) 5 months, (*B*) 2 years, 10 months, and (*C*) 4 years of age.

congenital heart lesion. Involvement of the *skeleton* alone seems to be compatible with long life, in some instances with little disability.

When the patient has involvement of *both* the lungs and bones (and perhaps other tissues also), the course seems to be inevitably progressive to death. Autopsy studies have shown angiomatous involvement of most of the viscera and chylous effusions in the body cavities. The effusions have in several instances been the presenting condition which has led to the establishment of a diagnosis. The nature of the fluid withdrawn by paracentesis varies from serosanguineous to frankly chylous. Several reports have mentioned progressive destruction of the bones over a period of time. Sometimes, biopsy of the involved bones was carried out, without arriving at a definitive diagnosis. In most instances of disseminated disease, the principal osseous involvement was in the bones of the thorax and shoulder girdle. This was true also in our patient.

ILLUSTRATIVE CASE

CASE III. A white male was first admitted at 16 days of age because of congenital defects which included harelip and cleft palate, exotropia, umbilical hernia, and hypospadias. During numerous subsequent admissions for repair of these defects, chest roentgenograms were ob-

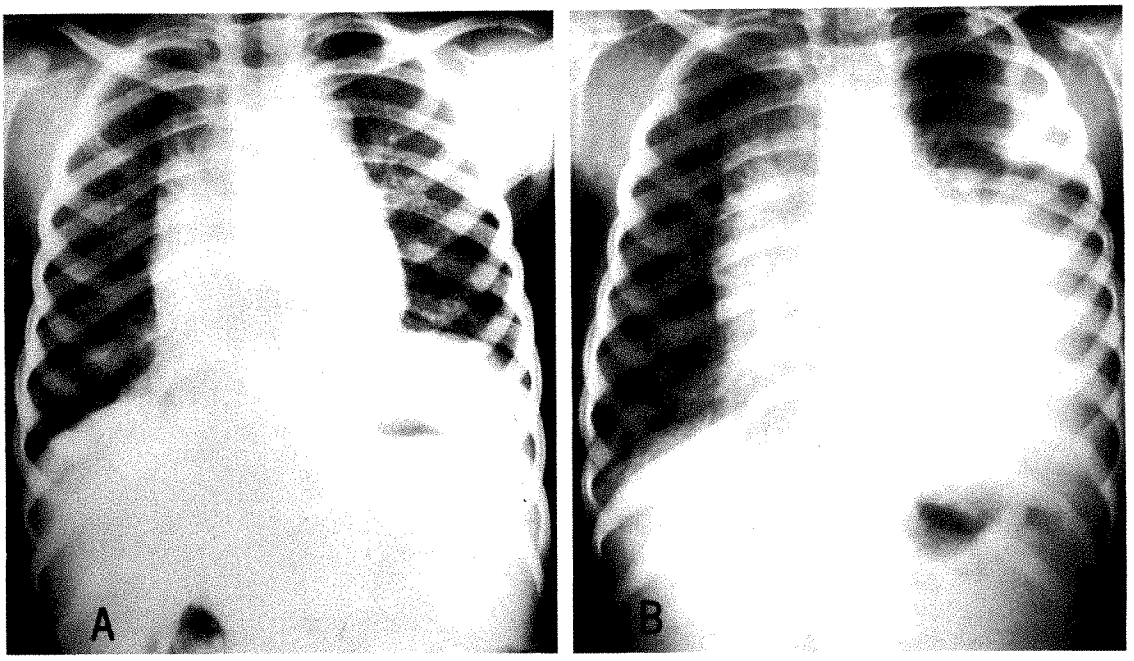


Fig. 6. Case III. (*A*) Roentgenogram made 2 weeks after Figure 5*C* showing an increased amount of left pleural effusion. The pleural fluid rapidly increased to the extent shown in *B*, made 18 days after *A*.

tained which showed a progressive prominence of diffuse markings in the lung fields and a gradual widening of the mediastinum (Fig. 5, *A–C*). At 4 years of age he presented with a left pleural fluid collection (Fig. 6, *A* and *B*). The fluid withdrawn by thoracentesis was the color and consistency of tomato soup. The white blood cell count was 8,000 per cu. mm., and the red blood cell count was 1,000,000 per cu. mm. Total protein was 5.7 gm. per cent and total lipid 1.9 gm. per cent. Cultures and cell blocks were negative for organisms and tumor cells. A mottled destructive process, which suggested malignant tumor, was seen in the scapulas and in the first two right ribs, but the boy was without pain or signs of toxicity. His family noticed, however, a bluish swelling across his shoulders and back, "like a spare tire," when he became cold while swimming.

A biopsy of the spine of the right scapula was carried out June 22, 1957. The histologic examination showed no remarkable abnormality of bone architecture except in one particular. Between the spicules of bone there were masses of fibroadipose tissue which in several sites contained large, empty cystic spaces lined by a fine rim of fibrous tissue. The significance of these cystic spaces was not apparent and no frank evidence of a neoplastic process was found.

Physical examination revealed a fullness of the suprasternal notch. This was aspirated, but no diagnosis could be made from the tissue obtained by this needle puncture. A thoracotomy was performed on June 28, which revealed a diffusely infiltrating dark reddish tumor of the mediastinum. The pathologic diagnosis was lymphangioma.

Radiation therapy was directed to the mediastinum, 2,100 r from a 220 kv. machine in 26 days, but apparently had no effect in slowing the formation of the chylothorax, and thoracenteses were required about once a week.

On August 23, 1957, another thoracotomy was performed and the thoracic tumor was largely removed. The pathologist described the tumor as being composed of irregular endothelial-lined spaces of variable size, with lymphoid aggregates and numerous small interspersed accumulations of inflammatory cells (mainly plasma cells). Again, a diagnosis of lymphangioma was made. Following this operation there was no recurrence of the chylothorax. The patient felt very well.

Toward the end of 1957, he commenced to have fever and severe headaches. Roentgenograms showed progressive destruction of the bones at the shoulder girdle and in the neck (Fig. 7). The skull also showed "bubbly" defects in the occipital bone (Fig. 8, *A–D*). Be-

cause there was so much loss of bone from the cervical spine that the patient was considered to be in danger of vertebral collapse which might damage his spinal cord, a cervical vertebral fusion was attempted June 25, 1958. When the immobilizing cast was removed 10 weeks later, the cervical defects were even more conspicuous than preoperatively, and the posterior graft had not fused (Fig. 9 and 10). Two days after the cast was removed, the patient quietly expired.

Autopsy was limited to the thorax and showed extensive involvement by abnormal endothelial cystic tissue of the pleurae, mediastinum (Fig. 11), periaortic lymph nodes, cervical and thoracic vertebrae, and scapulae. The heart revealed a subacute interstitial myocarditis. There was an organizing fibrinous pleuritis and pericarditis. The terminal event appeared to be the result of bilateral bronchopneumonia.

Comment. The clinical course in this patient was much like that in the patient of Cohen and Craig.[5] They pointed out that bone biopsies were difficult to evaluate because the friable nature of the lesion permitted the removal of only small fragments. In addition, our patient was subjected to radiation therapy, but without success in controlling the formation of chylothorax.

DISCUSSION

Angiomatosis is the term recently used by Koblenzer and Bukowski[16] to describe the disease, thereby avoiding the issue as to whether the condition is essentially a hemangiomatosis or a lymphangiomatosis. "Hamartomatous hem-lymphangiomatosis" is their all-inclusive subtitle. They feel that the disease is essentially an abnormality of the lymphatic system in which extensive venolymphatic communications occur. The main features in their description of the process are the osteolytic lesions, visceromegaly, cutaneous hemangiomas or lymphangiomas, and massive effusions into any body cavity (the fluid being chylous, sanguineous, or mixed).

SUMMARY

The clinical course of a disease causing widespread proliferation of the endothelial

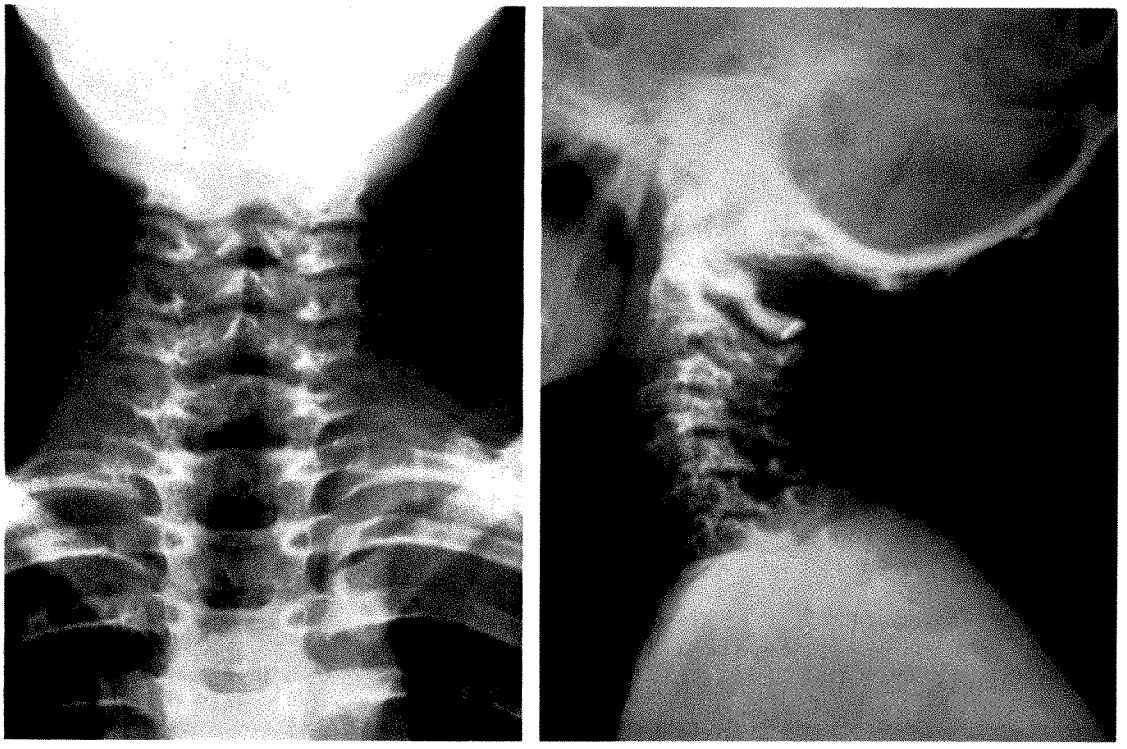


FIG. 7. Case III. The cervical spine at $4\frac{1}{2}$ years showed a severe "moth-eaten" destruction. A vesicular appearance also is visible in the occipital bone of the skull.

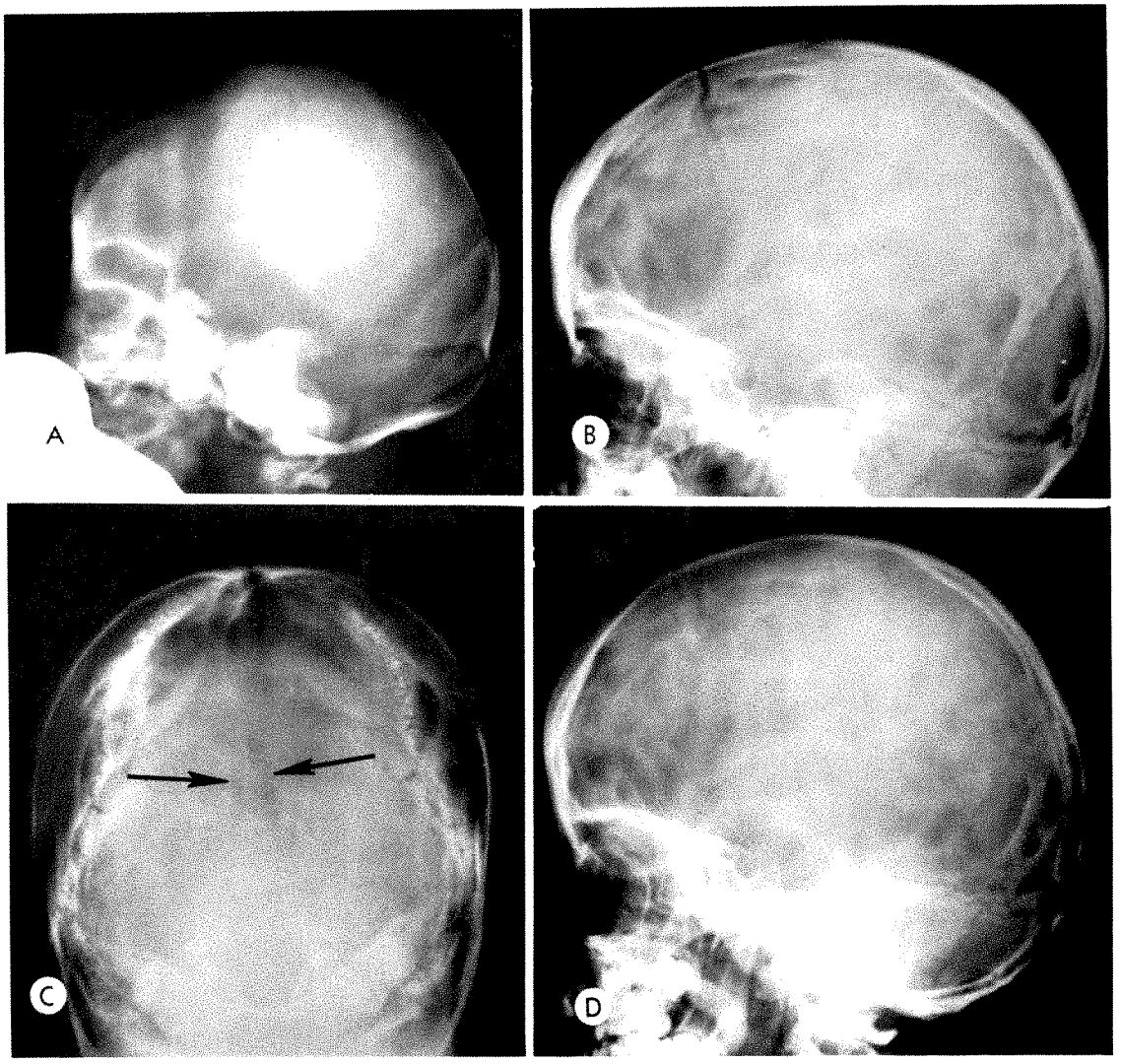


Fig. 8. Case III. (*A*) Skull at 16 days, (*B* and *C*) 4 years, and (*D*) 4½ years of age. *B* and *C* show prominent diploic bulges in the occipital bone (arrows in *C*). In *D* the occipital diploic space has enlarged and become continuous so that it separates the inner and outer tables.

tissues and dilatation of their channels varies markedly, but usually falls into one of three categories. A case is reported to illustrate each category.

Lymphangiectasis limited to the pulmonary tissues is a serious defect which heretofore has been described only in fetuses and newborn infants. That there may be milder forms of the anomaly and that they may be associated with congenital heart disease is suggested by our report and by Giammalvo.[7]

Lymphangiectasis confined to the osseous system has been described as producing over a period of years a condition which spreads in one or multiple bones but which in many instances seems self-limited. "Massive osteolysis" seems to best describe the process, whether it be of lymphatic or vascular origin. The presented case of osseous lymphangiectasis is the earliest one reported in the literature, and Figure 4, showing the patient's arm, is published for the first time.

The diffuse or multiple-system form of the disorder is invariably fatal but may run a prolonged and insidious course before the diagnosis becomes evident. As with the

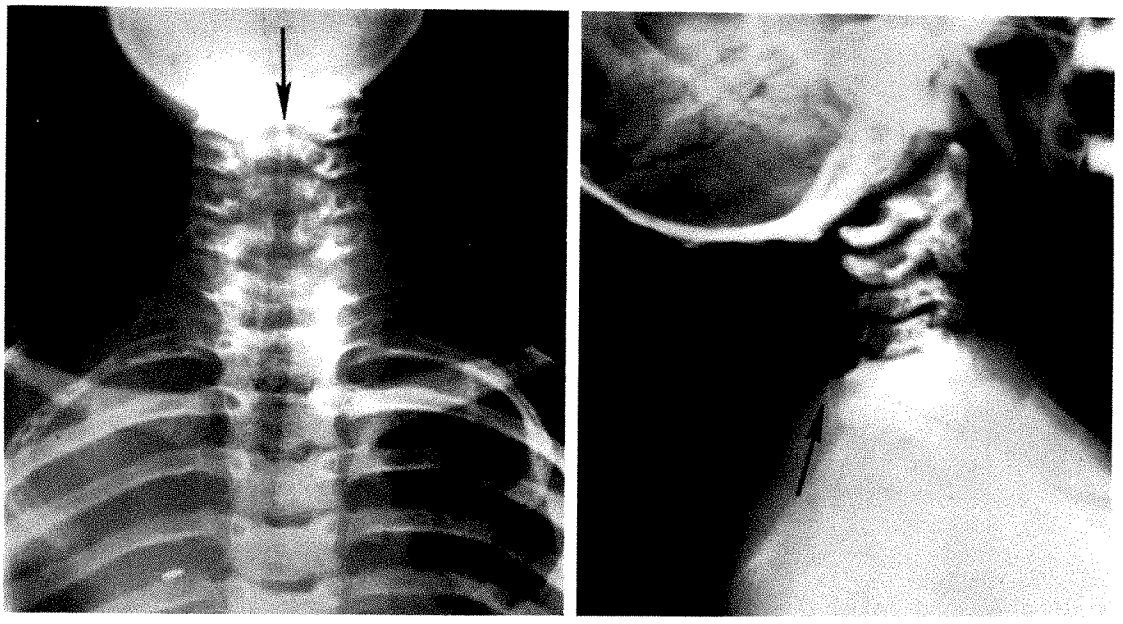


FIG. 9. Case III. Cervical spine 10 weeks after unsuccessful attempt at posterior fusion. Arrows point to unfused bone graft. The first ribs are so much resorbed as to be almost unidentifiable. The mottled nature of the bone destruction is best visualized in the right second rib.

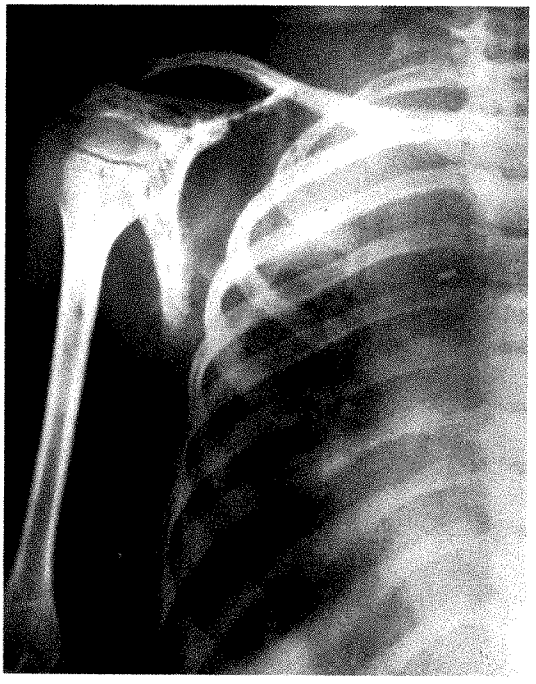


FIG. 10. Case III. Detail of right side of shoulder girdle from a chest roentgenogram made at 5 years, 4 months of age. A honeycomb type of destruction is evident in the scapula and ribs. Pleural effusion, which had previously involved only the left side of the thorax, can now be seen on the right side.

other forms of lymph- or hemangiectasis, there appears to be no neoplastic component, but rather a hamartomatous condition which advances inexorably in a mechanical progression involving multiple body tissues, but especially those of the upper trunk.

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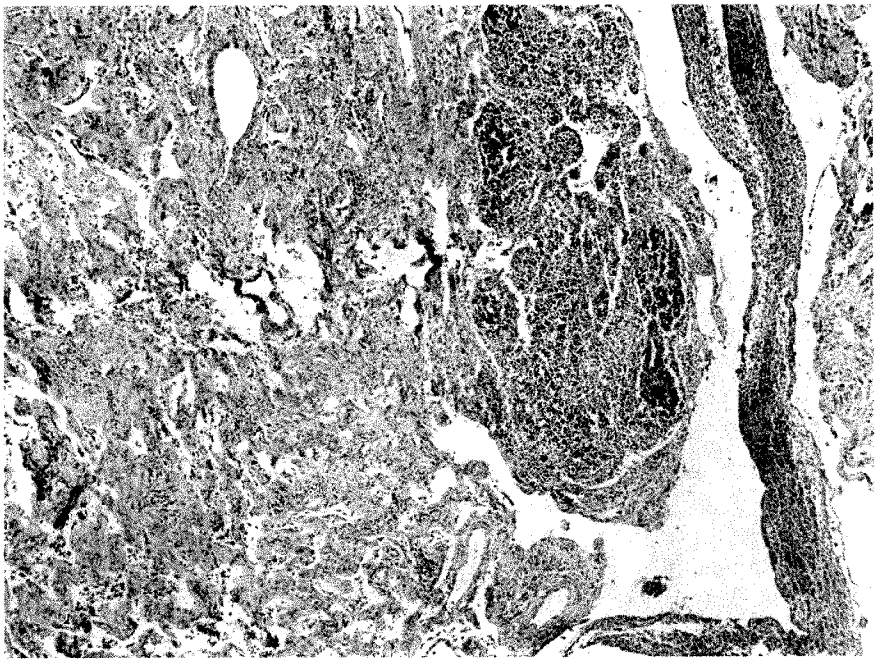


FIG. 11. Case III. Section from mediastinum at autopsy (H. & E., ×40). The left half of the field shows rather solid tissue of lymphatic origin. In the right half are a lymph node and a large endothelium-lined lymph space.

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CYSTIC ANGIOMATOSIS OF BONE: MANIFESTATION OF A HAMARTOMATOUS DISEASE ENTITY*

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NEW YORK, NEW YORK

THE term, cystic angiomatosis of the skeletal system, was suggested by Jacobs and Kimmelstiel[5] in 1953, to describe a condition characterized by multiple cystic lesions in the skeleton, considered by them to be due to numerous scattered hemangiohamartomata. More recently, Gramiak, Ruiz and Campeti[2] have used the same designation in describing what they consider to be a rare form of generalized hemangiomatosis which produces cystic changes in bones. We have recently seen a patient with bone changes identical to those described and illustrated by the above authors. A correct roentgen diagnosis was made prior to biopsy. A review of 10 similar cases of osseous angiomatosis previously reported in the English language literature, however, suggests rather strongly that the condition currently designated cystic angiomatosis of bone is, indeed, only one expression of a broader disease entity, the pathologic common denominator of which is a hamartomatous cystic vascular malformation. The following case is reported in order to illustrate the characteristic osseous roentgen features and to present what we consider to be a relatively benign form of a disease entity which may be manifested in all grades of severity including some which are lethal to the patient.

REPORT OF A CASE

A 14 year old Puerto Rican boy was admitted to the hospital because of malaise and fever of 1 week's duration. Three days prior to admission he developed a nonproductive cough, had several episodes of vomiting and complained of generalized muscle pains. On admission the temperature was 102° F., the pulse 82, and the respirations 40. Fine crepitant rales were heard at the bases of the lungs. The physical examination was otherwise entirely negative. Neither the spleen nor liver were palpable. The hemoglobin was 13.9 gm./100 ml. The white blood cell count was 10,250, with 76 per cent neutrophils, 16 per cent lymphocytes, 4 per cent monocytes and 4 per cent eosinophils. The erythrocyte sedimentation rate was 73 mm. in 1 hour. A clinical diagnosis of bronchopneumonia was made and the patient was sent to the department of radiology for a roentgen examination of the chest. This examination failed to demonstrate any pulmonary infiltrations but cyst-like areas of bone destruction were noted in the left fourth and seventh ribs and in the right seventh rib (Fig. 1). At the sites of involvement the bone was expanded and the cortices were thinned. A roentgen skeletal survey was suggested for further evaluation of the osseous abnormalities. After recovery from the respiratory infection, a bone survey showed multiple cyst-like areas of bone destruction involving the pelvis, skull, right scapula, both clavicles, the femora, the spine and the proximal phalanx of the left big toe (Fig. 2, 3 and 4). Laboratory tests made at this time yielded no clues to the nature of the bone lesions and, although Schüller-Christian disease and polyostotic fibrous dysplasia were considered in the roentgen differential diagnosis, it was suggested that the most likely diagnosis was cystic angiomatosis of bone. An attempt at needle aspiration of an iliac lesion was unsuccessful and an involved segment of the right seventh rib was then resected for pathologic study (Fig. 5). When the affected portion of this rib was exposed before resection, it showed a bluish discoloration with expansion of the bone contour and marked thinning of the cortex. A 3 inch section was resected. This was examined pathologically and was reported as showing cystic hemangioma.

Comment. There are 9 reports in the English language medical literature describing a total of 10 cases in which angiomatosis has involved bone in such a way as to pro-

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duce multiple cystic areas of destruction. In most of these cases, in addition to the bone changes, there have been lesions of a similar histopathologic nature involving various visceral organs and other soft parts (Table 1). Although the spleen, pleura and skin have been the most frequently involved soft tissues, involvement of the lungs, liver, kidneys, pericardium, thymus and retroperitoneal tissues has also been reported. In 1 case there was an associated congenital lymphangioma of the forearm and in another there were lymphangiomata of the neck and axilla.

PATHOLOGY

Although interpretation of the pathology found in the reported cases has varied, the pathologic descriptions have shown a remarkable similarity. The basic pathology, both in soft tissues and in bone, has consisted of cystic structures lined by a single layer of endothelium. In affected portions of bone, the marrow has been replaced by dilated extremely thin-walled cysts. Although in some instances the cysts were solitary, in most cases they have been multilocular and often communicating. These structures have been generally considered to represent dilated vascular spaces. The agreement has ended there, however, since various authors have described them as hemangiomata, lymphangiomata, lymphangiectasis, hemangiohamartomata and hamartomatous hem-lymphangiomatosis. Some of the cystic spaces have contained blood while others have contained a clear fluid. Cohen and Craig[1] in order to call attention to the difficulties involved in classification used the designation multiple lymphangiectasis of bone. So little is known

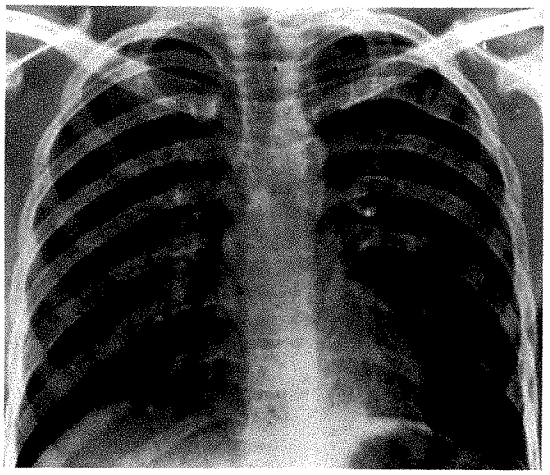


Fig. 1. There are multilocular cyst-like areas in the posterior portion of the left fourth rib (upper left arrow). The lesions are medullary and have expanded the bone and thinned the regional cortices by erosion of their inner aspects. There are multiple cyst-like areas in the left seventh rib in the axillary line (lower left arrow). Some sclerosis of the rib adjacent to the cysts suggests that there has been a previous fracture. A similar area of destruction may be noted in the right seventh rib posteriorly (right arrow). There are several small cystic lesions in the medial thirds of both clavicles. Most of these have peripheral rings of increased density but are difficult to delineate because of their small size and overlapping of bone and lung shadows.

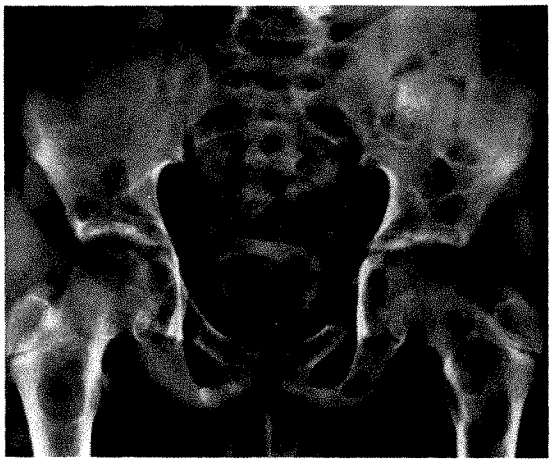


Fig. 2. There are multiple cyst-like areas of bone destruction in the iliac, pubic and ischial bones bilaterally. Similar lesions are scattered throughout the upper ends of the femora, including the right capital epiphysis. There are also lesions in the sacrum and a large one can be seen in the right transverse process of the fifth lumbar vertebra. Most of the lesions are sharply marginated and the majority of them have peripheral rings of increased density. There are poorly marginated lesions noted, however, in the neck and intertrochanteric region of the left femur. There is little or no trabeculation of the defects and a lesion in the left pubis appears to be cortical in origin. No reactive osteosis is noted adjacent to the areas of destruction. (Courtesy of Grune and Stratton, Inc., New York and London.)



FIG. 3. There are multiple small areas of osteolysis in the frontal and parietal bones of the skull. Some of these are well marginated with or without peripheral rings of increased density. Others are poorly circumscribed. One area (arrow) shows a honeycomb pattern much like that characteristically seen with hemangioma of the skull. There is erosion of the outer table in the region of the vertex. No areas of reactive osteosis are noted.

about the anatomy of lymph vessels in bone, however, that one approaches the diagnosis of lymphangioma or lymphangiectasis of bone with some caution. Willis[14] has emphasized the fact that hemangiomata and lymphangiomata need not exist as distinct malformations but may occur together. He places angiomata in the category of hamartomata which he has described as "tumor-like but primarily non-neoplastic malformations or inborn errors of tissue development, characterized by faulty mixture of tissues indigenous to the part with excess of one or more of these." It seems reasonable to consider these generalized vascular dilatations, whether hemangiomatous, lymphangiomatous or both as hamartomatous vascular malformations of a cystic nature. Clinical manifestations depend on the sites of involvement, the extent of dissemination and whether hemangiomatous or lymphangiomatous elements predominate. Koblenzer and Bukowski,[6] to avoid debate, have referred to this entity as hamartomatous hem-lymphangiomatosis.



FIG. 4. Lesions in the distal femoral shafts are elliptical with long axes parallel to the long axis of the shaft. They are sharply circumscribed with peripheral rings of increased density. At the lower end of the left femur, the lesion has partially eroded the adjacent cortex. Both lesions are eccentric. There is no reactive bone sclerosis.

CLINICAL FEATURES

When hamartomatous cystic vascular malformations are widespread and involve vital organs, the prognosis is poor. Parsons and Ebbs,[9] for instance, have reported the fatal case of a 14 year old girl in whom the liver, spleen, lungs, pleura, kidneys and skeleton were diffusely involved. Shennon,[12] Cohen and Craig[1] and Koblenzer and Bukowski[6] have also reported patients with diffuse visceral involvement who did not survive. When the lesions are predominantly osseous with little or no visceral in-

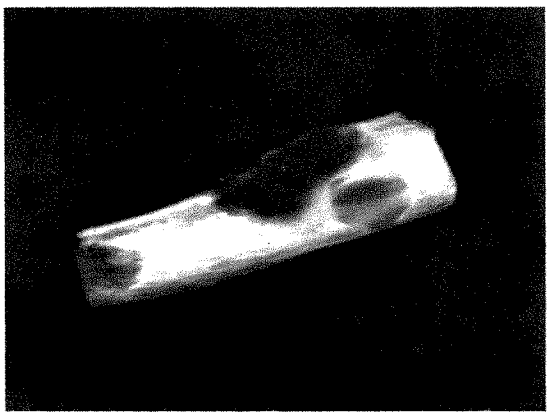


FIG. 5. Roentgenogram of resected rib shows clear cystic areas of destruction without trabeculation. The bone contour is expanded and the cortices are thinned.

Table I

Authors	Patient		Type of Involvement						Primary Clinical Manifestation	Diagnosis	Course
	Age When First Seen	Sex	Bone	Spleen	Viscera	Pleural Effusion	Skin	Other			
Parsons and Ebbs[9]	14	F	+	+	+		+		Swelling of sternum	Cavernous hemangiomatosis; generalized angiomatosis	Died
Shennon[12]	23	F	+	+	+	+	+		Cough; hemoptysis	Histologically nonmalignant angioma with metastases	Died
Harris and Prandoni[3]	20	M	+					Congenital lymphangioma of forearm	Lymphedema of forearm	Generalized primary lymphangioma of bones and congenital lymphangioma of forearm	Untraced
Cohen and Craig[1]	8	F	+	+	+	+			Limited motion of neck; fractures; paraplegia	Multiple lymphangiectasis of bone	Died
Ritchie and Zeier[10]	2½	M	+	+	+				Pathologic fractures	Hemangiomatosis of skeleton and spleen	Living 3 years later
Koblenzer and Bukowski[6]	4	F	+	+	+	+	+		Hemothorax	Hamartomatous hemlymphangiomatosis	Died
Jacobs and Kimmelstiel[5]	15	M	+						Fractures	Cystic angiomatosis of skeletal system	Untraced
	11	F	+					Lymphangioma of neck and axilla first noted at age 2	Soft tissue mass	Cystic angiomatosis of skeletal system	Untraced
Gramiak *et al.*[2]	15	M	+						Hemarthrosis; fractures	Cystic angiomatosis of bone	Living
Schopfner and Allen[11]	15	M	+						Pathologic fractures	Lymphangioma of bone	Living
Present Authors	14	M	+						None	Cystic hemangioma of bone	Living

volvement, however, the prognosis is good. Ritchie and Zeier[10] have described a 2½ year old boy with osseous and splenic involvement. The patient was seen primarily because of multiple fractures through involved areas of bone. This patient has been observed for 3 years following splenectomy and has maintained a state of good general health but during the period of observation has sustained 3 additional pathologic fractures. Harris and Prandoni[3] have described a 20 year old soldier with generalized skeletal involvement who also had a congenital lymphangioma of the forearm and brownish skin discolorations which were present since birth. His clinical complaints were limited to lymphedema of the affected forearm. Jacobs and Kimmelstiel[5] have reported 2 cases, in one of which generalized bone lesions were associated with lymphangiomata of the neck and axilla. In this instance the patient was seen because of the soft tissue masses. The bone lesions were discovered accidentally when routine roentgenographic examination of the chest revealed multiple cyst-like lesions in the ribs. In their other case only the skeleton was involved and the patient was seen because of recurrent fractures. Gramiak *et al.*[2] and Schopfner and Allen[11] have also reported

patients in whom lesions were confined to the skeleton. In both instances there was a history of fractures. In the case described by Schopfner and Allen, the bone defects were found incidentally on a chest roentgenogram made because of a clinical impression of pneumonia.

In the case reported in this paper the process is limited to the skeleton and is entirely symptomless. The lesions were discovered fortuitously on a roentgen examination of the chest made to confirm a clinical impression of pneumonia.

ROENTGEN FINDINGS

The roentgen bone changes in this condition are striking. Usually, they are so characteristic that a correct diagnosis can be made on the basis of the roentgen appearance alone. In all cases multiple bones have been affected. In every instance the skull, pelvis, ribs and femora have been involved. Involvement of one or both humeri has been demonstrated in 9 of the 11 cases and the scapula has been affected in 7. Slightly less often, the vertebrae and tibiae have been the sites of demonstrable lesions. More infrequently, lesions may be noted in the other long bones, the clavicles and sternum. In only 1 case have the short tubular bones of the hands or feet been mentioned as being involved but in most reports there is no indication that these parts were examined roentgenographically. It will be noted that in the current case there was a small cystic lesion in the proximal phalanx of the left big toe.

The similarity in roentgen appearance of the skeletal changes in all cases is striking and the pattern of bone change provides a strong basis for specific roentgenologic diagnosis.

The basic roentgen feature is that of multiple cyst-like areas of bone destruction. These usually show a peripheral ring of increased density. Some of them, however, may be sharply marginated but without a dense periphery and, occasionally, there may be little or no margination, the lesion gradually blending with the surrounding normal bone (Fig. 2 and 3). A peripheral ring of density is more characteristic of the lesions, however, and most of them will show this type of margination. Lesions tend to involve the medullary portion of the long bones and may be centrally or eccentrically situated but occasionally a cystic lesion may be cortical in origin (Fig. 2). Frequently, shaft lesions tend to be oval in shape, their long axis running parallel to the long axis of the bone within which they are located but some of them may be round or, rarely, irregular in shape (Fig. 4). The expanded cysts tend to thin the overlying cortex, sometimes to a considerable degree (Fig. 1, 4 and 5). While characteristically the area of destruction is uniform, an occasional lesion may show fine trabeculation and in rarer instances there may actually be a soap-bubble or honeycomb pattern (Fig. 3). Significant reactive osteosis adjacent to a lesion is unusual and periosteal reaction has not been noted. Where there has been a pathologic fracture, however, there may be some periosteal new bone formation or some residual osteosclerosis (Fig. 1). The epiphyses, metaphyses and shafts may be sites of involvement. In the ribs the cystic areas show a strong tendency to expand the bone, thinning the adjacent cortices and sometimes causing pathologic fractures. In the vertebrae and skull, lesions tend to be round and cystic without the vertical striations or peculiar honeycomb appearance usually seen in hemangiomata of these bones.

In several instances where patients have been observed over a period of time new lesions have been noted to develop and some lesions have been seen to enlarge.[1,3]

DISCUSSION

It can be seen from the ages listed in Table 1 that the age distribution in reported cases has ranged between $2\frac{1}{2}$ and 23 years. These figures represent the ages of the patients when they were first seen by

the authors who reported them. Review of the histories, however, clearly indicates that, in most cases, there were clinical manifestations of the condition at much earlier ages. It appears, therefore, that the onset of this condition usually occurs sometime in childhood. That it is not present at birth need cause no surprise since, according to Willis,[14] hamartomata, more than any other class of malformation, show the phenomenon of delayed appearance. Analogous to this is the late appearance of manifestations of some inborn errors of metabolism. The fact that in some cases the lesions have been observed to increase in size is more likely due to canalization and distention of already existing malformations by blood or lymph than to actual proliferative growth.

In all of the cases reviewed in this paper, the skeleton was involved by multicentric cystic areas of destruction, but it is possible that within this disease entity there may be instances in which only the soft parts are involved and the skeleton is unaffected. Miller *et al.*,[7] for instance, have described a fatal case of chylopericardium associated with an extensive hamartoma of the intrathoracic lymphatic channels without osseous lesions, and Hecht and Korb[4] have reported a 5 month old infant with diffuse lymph-hemangiomatosis of the skin, skeletal muscle, intestine, diaphragm, bladder, kidney and soft tissues of the neck. The patient also had a chylous ascites, but there was no bone involvement.

It is our feeling that when the bones are involved the appearance of the osseous roentgen changes are sufficiently characteristic to suggest the diagnosis, and when perplexing soft tissue manifestations are also present the roentgenographic features demonstrable in the bones may suggest the nature of the visceral abnormalities.

The literature abounds with reports of angiomatosis of one kind or another. The term has been used to include telangiectasia, hamartoma and actual neoplasia. It is possible, however, to separate the cases listed in Table 1 from this large heterogeneous group. Such cases all show multicentric cystic foci of bone destruction and similar histopathologic findings.

Of interest and practical importance is the fact that in almost all cases biopsies of bones other than ribs have been inconclusive. This is probably related to the ease with which an adequate section of involved bone may be obtained from a rib and the difficulty experienced in obtaining such a section from other bones. It is considered, therefore, that excision of an affected portion of rib is the procedure of choice in establishing a pathologic diagnosis. Blood chemistries and other laboratory procedures yield no clues to the nature of the condition and the great value of a correct interpretation of the roentgen skeletal changes is apparent.

DIFFERENTIAL DIAGNOSIS

The conditions most apt to be considered in the differential diagnosis of cystic angiomatosis of bone are histiocytosis X, polyostotic fibrous dysplasia and hyperparathyroidism. The osseous defects in histiocytosis X tend to have a distribution similar to those of angiomatosis but the early lesions, although well circumscribed, usually do not have a peripheral ring of increased density and healing or treated lesions are usually associated with some degree of reactive osteosclerosis.[8] Periosteal reaction is not uncommon in histiocytosis X but has not been observed in cystic angiomatosis. When cystic angiomatosis involves the liver and spleen, there may be a clinical resemblance between the two conditions requiring more detailed analysis. The so-called cystic type of lesion in fibrous dysplasia may resemble the bone lesions of cystic angiomatosis, but frequently close inspection of such defects will reveal a ground-glass hazy density due to fibro-osseous tissue within the cyst-like defect of fibrous dysplasia. Where there is diffuse involvement of the skeleton there are apt to be portions of bone which are undertubu-

lated or locally expanded and a variety of osseous deformities including bowing, acetabular protrusion and coxa vara may be seen. In hyperparathyroidism granular decalcification of the skull, subperiosteal resorption of bone, generalized decalcification and disarrangement of the trabecular architecture are important diagnostic features.

SUMMARY

A case of cystic angiomatosis of bone is described in a 14 year old Puerto Rican boy. Ten previously reported cases showing a similar roentgen pattern of skeletal involvement and similar pathologic findings are reviewed and tabulated. In some of these the pathologic process was limited to the skeleton, but in others the bone changes were associated with lesions of similar pathology in various soft parts. The basic pathology in both soft tissue and bone consists of unilocular or multilocular cyst-like dilated vascular spaces. These show no features of neoplasia and are considered to be hamartomatous vascular malformations. It is suggested that cases showing only generalized involvement of bone by lesions of this type represent one manifestation of a broader hamartomatous disease entity which may be manifested in all grades of severity, ranging from those which are lethal to those which are benign and asymptomatic.

The roentgen pattern of bone change is characteristic and usually permits a specific roentgenologic diagnosis. As no other laboratory or clinical findings short of biopsy provide a significant clue to the nature of the condition, the importance of correct roentgen interpretation of the skeletal defects is evident.

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DETECTION OF METASTATIC CANCER TO BONE BY SCINTISCANNING WITH STRONTIUM 87m*

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THE use of radioactive strontium as a tracer of calcium metabolism in malignant disease of bone can be said to have been ushered in with the report of Treadwell and associates in 1942 concerning the utilization of strontium 89, a pure beta emitter, in autoradiographic studies of primary bone tumors.[19] The results of these investigations indicated that radioactive strontium was deposited in areas of active osteogenesis both in normal bone and in osteogenic sarcoma.

More recently, several groups of investigators have employed Sr^{85}, a pure gamma emitter, to trace certain aspects of calcium metabolism in man.[2,6,17] It has been shown that although quantitative differences exist in gastrointestinal and renal handling of the two elements, there is much less discrimination by bone.[6] Bauer and Wendeberg were successful in demonstrating increased strontium accretion by means of external counting in bones damaged by fracture, infection, tumor and Paget's disease;[3] and Gynning *et al.* found that increased Sr^{85} deposition in bone could precede visible roentgenographic changes in patients with metastatic breast cancer.[8] This finding indicated that local changes in radiostrontium metabolism might be a more sensitive index of the presence of bone metastases than the roentgenogram, in which calcium content alterations of 30 to 50 per cent must occur before visible changes become apparent.[1,16]

In 1961, Fleming and associates demonstrated that the spatial distribution of Sr^{85} in the human could be displayed by means of photoscanning.[7] Sklaroff and Charkes employed Fleming's technique and showed that the scintiscanning method is capable of demonstrating metastatic tumor to bone prior to observable roentgenographic changes, as proven by bone biopsy.[4,13,14,15]

Despite these salutary findings, scanning with Sr^{85} has several disadvantages: (1) Because of its long physical half-life (64 days) and its slow turnover rate in bone (biologic half-lives of 800[18] and 843 days[5] have been recorded), frequent repetition of the scan in order to evaluate response to therapy is not feasible; (2) as a consequence of the long effective half-life, the estimated radiation dose to bone of 4.6 rads per 100 μc injected[7] (a lower integral dose is probably more likely—see Table 1) has limited the injected dose to 100 μc or less. This in turn results in suboptimal resolution and long scanning times due to poor counting statistics; and (3) Atomic Energy Commission approval has been granted solely for study of malignant diseases of bone.

It would be desirable to extend these studies to encompass nonmalignant bone disorders including those of children, particularly with a strontium isotope of short half-life and no particulate emission so that relatively high doses could be safely administered at frequent intervals to assess the osteogenic response to therapy.

Strontium 87m satisfies these conditions (Table 1), and was therefore proposed as a substitute for Sr^{85} by Myers in 1960.[10] It decays by emission of a single gamma photon of 0.388 mev. to stable Sr^{87}, and has a half-life of 2.8 hours. There is no associated particulate nuclear emission and, although the internal conversion coefficient is 0.22, the radiation dose to the whole body and to bone is less than 1 per cent that of Sr^{85}.

We have recently obtained a series of Sr^{87m} generators from Brookhaven National Lab-

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oratories and have evaluated this isotope in a group of 16 patients, in 6 of whom Sr^{85} scans were also performed for comparison. Photoscans made with the two isotopes were identical. Just as with Sr^{85}, Sr^{87m} scans may reveal the presence of tumor in bone prior to observable roentgenographic changes, as proven by bone biopsy.

METHODS

Sixteen patients have been scanned to date with Sr^{87m}, including 7 patients with metastatic cancer to bone and 4 with primary bone tumors. In 6 patients, Sr^{85} scans were performed for comparison.

Sr^{87m} is obtained as an yttrium 87-strontium 87m generator, which is manufactured by bombardment of a rubidium target with 35 mev. alpha particles in the Brookhaven National Laboratories cyclotron. The Y^{87} (half-life 80 hours) is fixed on a Dowex-1 column, and the Sr^{87m} is obtained by "milking" the "cow" with 0.005 per cent citric acid solution at pH 5.5. Under these conditions, the yttrium 87 citrate remains on the column and the carrier-free Sr^{87m} passes through.[12] The solution is sterilized by passage through an ultrafine fritted glass filter into a sterile flask under negative pressure. Hypertonic (5 per cent) saline is added to bring the solution to isotonicity, and additional 0.9 per cent N saline is flushed through the filter to wash off adsorbed Sr^{87m} atoms. The isotonic solution is then agitated and an aliquot taken for counting prior to intravenous injection.

Counting efficiency for Sr^{87m} was determined by interpolation on a curve constructed from a plot of efficiency versus photon energy for the monoenergetic gamma emitting isotopes Cs^{137}, Sr^{85}, Au^{198}, Cr^{51} and Hg^{203}, and found to be 0.45 (corrected for interval conversion). Thus 1 μc of Sr^{87m} gave 781,000 cpm. Frequent standardization checks against a Cs^{137} source were made.

Two identical detectors and instrumentation systems consisting of a 2×2 inch NaI (Tl) crystal housed in a flat-field collimator, pulse height analyzer, count rate meter and strip chart recorder* were positioned over the precordium and right knee respectively, to measure blood disappearance rate and exchangeable strontium in bone following intravenous injection.

Scans were performed with the Picker Magnascanner,† utilizing the 19-hole collimator and pulse-height analysis.

RESULTS

The rate of accumulation of Sr^{87m} by the knee follows the general formula $A=A_0e^{kt}$ (Fig. 1). Measurements in 8 patients revealed a mean uptake $T\frac{1}{2}$ of 9.9 ± 3.5 min-

* Picker Twinprober.
† Picker X-Ray Corp., White Plains, N. Y.

TABLE I

COMPARISON OF STRONTIUM 85 AND STRONTIUM 87M

	Sr^{85}	Sr^{87m}
Gamma emission in kev. (% of disintegrations)	513 (100%)	388 (100%)
Beta emission	none	none
Internal conversion coefficient	0.8%	22%
Half-life	64 days	2.8 hours
Γ Dose rate constant in r/mc/hr. @ 1 cm.	3.0	1.65
Whole body dose per 100 μc injected*	680 millirads	2.2 millirads
Bone dose per 100 μc injected*	1,590 millirads	9.9 millirads
Production method	(1) Cyclotron; or (2) reactor: $Sr^{84}(n,\gamma)Sr^{85}$	Cyclotron: Y^{87} (E.C.) Sr^{87m}

* Injected into Standard Man (70 kg.), assuming 50 per cent retention for Sr^{85} and 100 per cent for Sr^{87m}, and biologic data of ICRP Report No. 2.[11] Equations of Hine and Brownell[9] were used for calculation of beta dose from electron capture (Sr^{85}) and conversion electrons (Sr^{87m}), and for integral dose. Geometry factor (g) = 41.6 (cylinder of radius 5 cm., volume 7,000 cc.) was used for calculation of gamma dose to bone.

utes (±1 S.D.) (Tables II and III). From these considerations it was found desirable to begin scanning approximately 45 minutes following injection so that 90–95 per cent of the Sr^{87m} to be deposited would have done so, and yet physical decay would be minimal.

Urine was collected at the end of the scanning procedure in 3 patients and was analyzed for Sr^{87m} by measurement of an aliquot in a well scintillation counter. Appropriate correction for decay was made. In all 3 patients, less than 4 per cent of the administered dose was recovered in $2\frac{1}{2}$ hours (Table II).

A normal Sr^{87m} scan of the pelvis is shown in Figure 2. In all 7 patients with metastases, abnormal areas of osteoblastic activity were noted on the Sr^{87m} scan (Table II; Fig. 3*A*). In 6 patients more extensive disease was visualized on the scan than was apparent on the corresponding roentgenogram (Fig. 4), and bone biopsies taken from scan-positive areas in 5 patients revealed the presence of tumor cells in all. In 2 patients bone biopsies from scan-positive areas revealed metastatic carcinoma although the roentgenograms were interpreted as normal.

In 6 patients to whom Sr^{85} was also administered, identical photoscans were obtained (Fig. 3 and 4). Two patients with eosinophilic granuloma of bone and roentgenographic changes showed no evidence of abnormal osteogenic activity on scan. In 1 patient with osteogenic sarcoma marked Sr^{87m} accumulation was noted (Fig. 4*A* and 5).

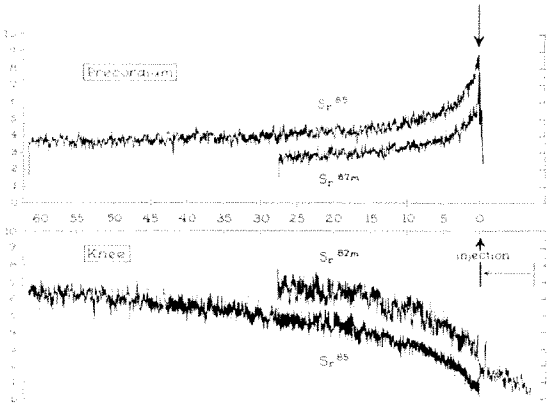


FIG. 1. Precordial disappearance (above) and knee uptake (below) of Case 10, injected successively with Sr^{87m} (54 μc) and Sr^{85} (20 μc), uncorrected for decay. Abscissa in minutes. Knee uptake follows function $A = A_0e^{kt}$; Sr^{87m} $T\frac{1}{2} = 9.0$ min. (corrected for physical decay), Sr^{85} $T\frac{1}{2} = 15.0$ min.

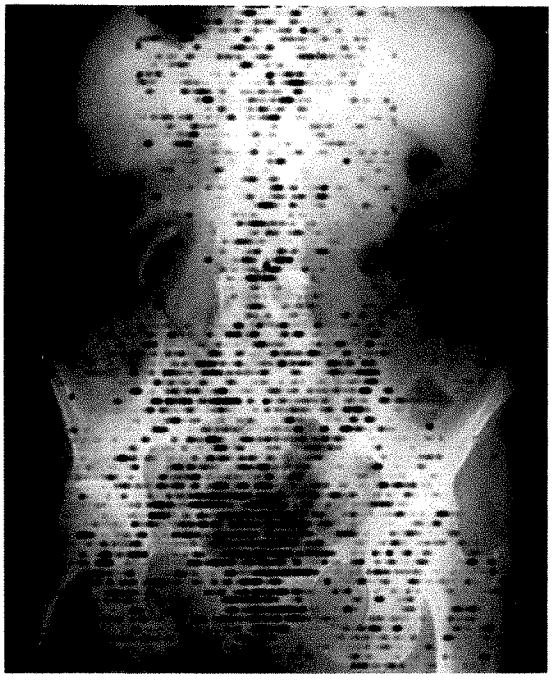


FIG. 2. Scan of 61 year old female with back pain (Case 14), begun 55 minutes following injection of 244 μc of Sr^{87m}. Note normal isotope content of lumbar spine and pelvis, particularly in articular surfaces (sacroiliac joints, femoral heads and acetabula), and in urinary bladder and urethra. Localization is identical to that found with Sr^{85}.[11,12,13]

DISCUSSION

These findings indicate that photoscanning with Sr^{87m} can detect metastatic cancer to bone even prior to observable roentgenographic changes. Sklaroff and Charkes showed that the photoscanning technique employing Sr^{85} can also demonstrate tumor deposits in bone despite normal roentgenograms.[4,13,14,15] Their results in 90 patients were substantiated by bone biopsy in 14 and confirmed by well-scintillation counting of the bone specimens. Resolution of the system was determined by phantom studies and by evaluation of scans of fracture sites,

TABLE II

RELEVANT DATA IN 16 PATIENTS INJECTED WITH STRONTIUM 87M

Case	Age	Sex	Tumor (site of origin)	μc Sr^{87m} injected	Knee uptake $T_{\frac{1}{2}}$ (min.)	Urinary excretion (min.)	Sr^{85} scan	Sr^{87m} scan	Roentgenogram	Biopsy*	Remarks
1	18	M	Eosinophilic granuloma (rib)	13	—	—	—	negative	positive	positive	
2	66	F	Eosinophilic granuloma (ilium)	105	—	—	—	negative	positive	positive	
3	67	M	Carcinoma (prostate)	177	—	—	positive	positive	positive	positive	Scan more extensive
4	43	F	Carcinoma (breast)	156	—	2.2% (155)	positive	positive	positive	—	Scan more extensive
5	66	F	Carcinoma (breast)	130	—	—	positive	positive	positive	positive‡	Scan more extensive
6	59	M	Normal (knee pain)	401	12.5	—	—	negative	negative	—	
7	86	M	Carcinoma (prostate)	470	6.0	—	—	positive	negative	—	Pain relieved by roentgen therapy of scan-positive area
8	76	F	Carcinoma (breast)	206	—	—	positive	positive	positive	—	Fig. 3, *A* and *B*
9	17	F	Malignant teratoma (ovary)	160	—	—	—	positive	positive	—	
10	9	F	Ewing's tumor (spine)	54	9.0†	—	positive	positive	positive	positive	Fig. 1
11	68	M	Osteomyelitis (L 1, 2)	1,568	17.0	—	—	positive	positive	—	Scan more extensive
12	69	F	Carcinoma (? origin)	494	9.0	3.6% (150)	—	positive	positive	positive	
13	60	M	Carcinoma (lung)	102	—	—	—	negative	negative	—	
14	61	F	Normal (back pain)	244	10.0	0.51% (140)	—	negative	negative	—	Fig. 2
15	46	F	Carcinoma (cervix uteri)	129	6.5	—	—	negative	negative	—	
16	13	F	Osteogenic sarcoma (tibia)	193	9.5	—	positive	positive	positive	positive‡	Fig. 4, *A-C* and 5

* Histopathologic examination of scan-positive area.
† Sr^{85} knee uptake $T_{\frac{1}{2}}$ = 15.0 min.
‡ Biopsy of scan (+), roentgen (−) area.
— = Not done.

and was found to be satisfactory. In most patients, the Sr^{85} scan correlated well with the roentgenogram, but in 11 the scan was positive and the roentgenograms negative. Furthermore, in many of the roentgenographically positive cases, the scan revealed more extensive disease than was visualized on the roentgenogram, and this was confirmed by bone biopsy in all patients so examined. Both osteolytic and osteoblastic lesions were positive on scan.

The present study indicates that photoscans produced by Sr^{87m} are identical to those made with Sr^{85} (Fig. 3, *A* and *B* and 4, *A* and *C*). In 6 patients scanned with both isotopes no difference in isotope deposition was noted, either in normal or in abnormal areas of osteogenesis. In 5 patients, bone biopsies of scan-positive areas were positive for tumor when examined microscopically, and in 6 patients the scan revealed more extensive changes than were apparent on the roentgenogram. In 2 patients bone biopsies of scan-positive areas revealed tumor although corresponding roentgenograms were interpreted as normal. It is apparent, therefore, that photoscanning with Sr^{87m} may reveal the presence of tumor in bone before visible roentgenographic changes are appreciated, and there are no essential differences between Sr^{87m} and Sr^{85} in this respect.

TABLE III

KNEE UPTAKE OF STRONTIUM 87M AND STRONTIUM 85

Isotope	No. of cases	Mean dose (μc)	Mean knee uptake $T_{\frac{1}{2}}$ (min.)	±1 S.D.‡
Sr^{87m}*	8	443	9.9	±3.5
Sr^{85}†	16	50	14.9	±6.3

* Carrier-free.
† Stable carrier added.
‡ Standard deviation.

Comparison of the knee uptakes of Sr^{87m} and Sr^{85} (Table III) reveals that quantitative differences do exist, however, in the rate of isotope deposition. This discrepancy may be due either to a dose effect or to the pres-

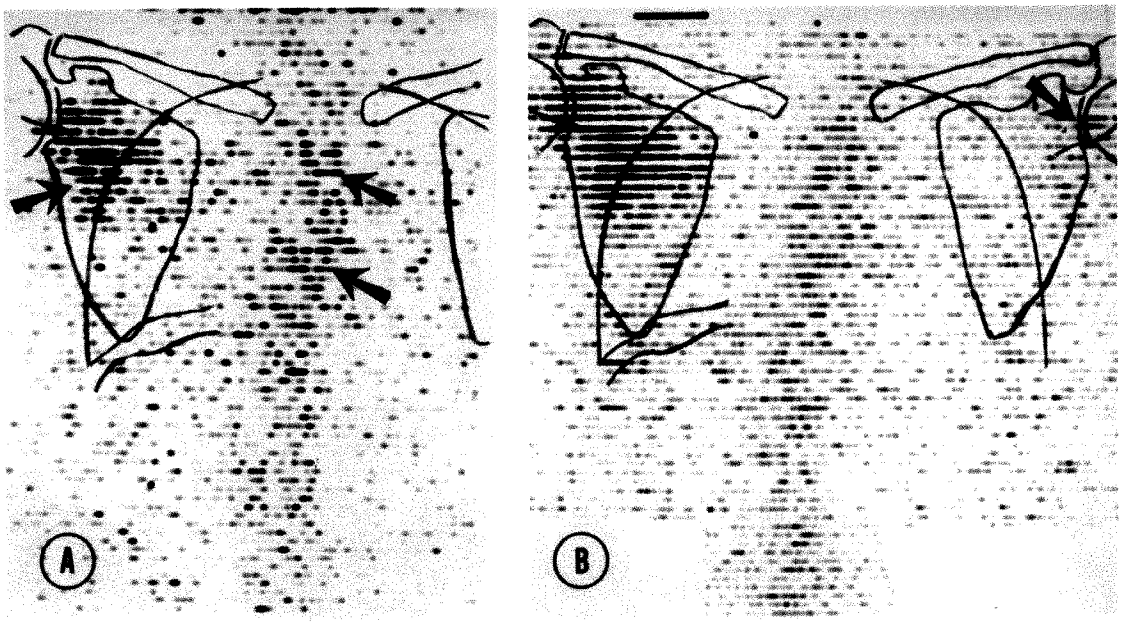


FIG. 3. Posterior chest scan of 76 year old female (Case 8) who developed multiple metastases to bone 30 months following right mastectomy for carcinoma. (*A*) Sr^{87m} scan shows extensive uptake in right scapula (arrow). (*B*) Identical localization is seen with Sr^{85}, injected 8 days later. Metastasis to scapula had been overlooked on initial chest roentgenogram. Normal left scapula is visualized on Sr^{85} scan. Note slight but normal increase in strontium deposition in left glenoid fossa and humeral head (arrow), and abnormal uptake in mid-dorsal spine (arrows). Metastases to D 4 and D 7 were seen on roentgenogram.

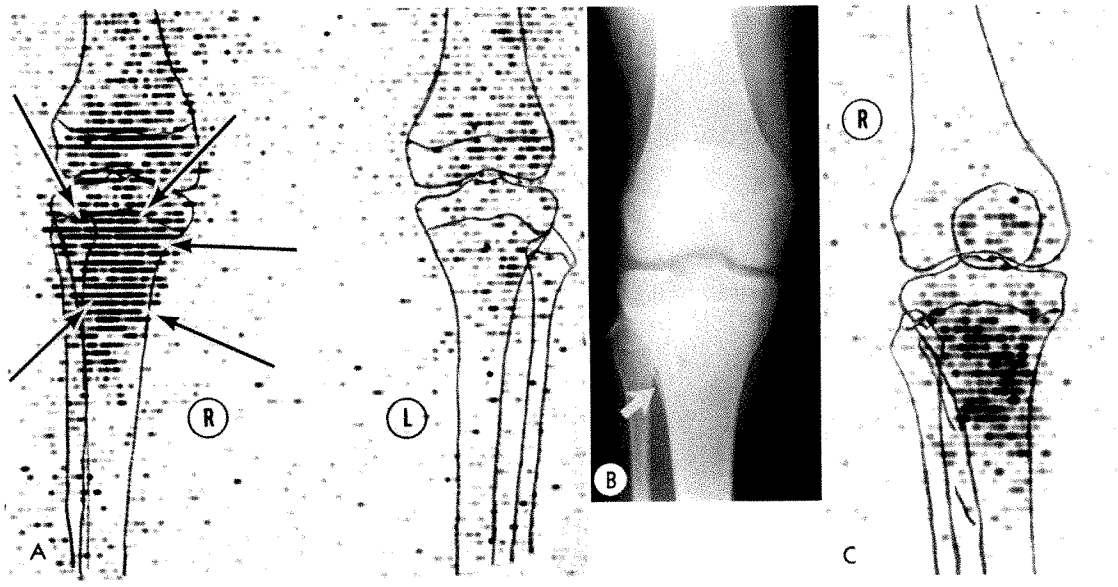


FIG. 4. (*A*) Sr^{87m} scan of legs of 13 year old female with osteogenic sarcoma of right tibia (Case 16). Note extensive strontium uptake (arrows) although only roentgenographic evidence of disease (*B*) was elevated periosteum (arrow). (*C*) Sr^{85} scan made subsequently shows similar localization. Notice increased strontium accumulation in femoral and tibial condyles and patella as compared with shaft on both scans, a normal finding.[5,6,12]

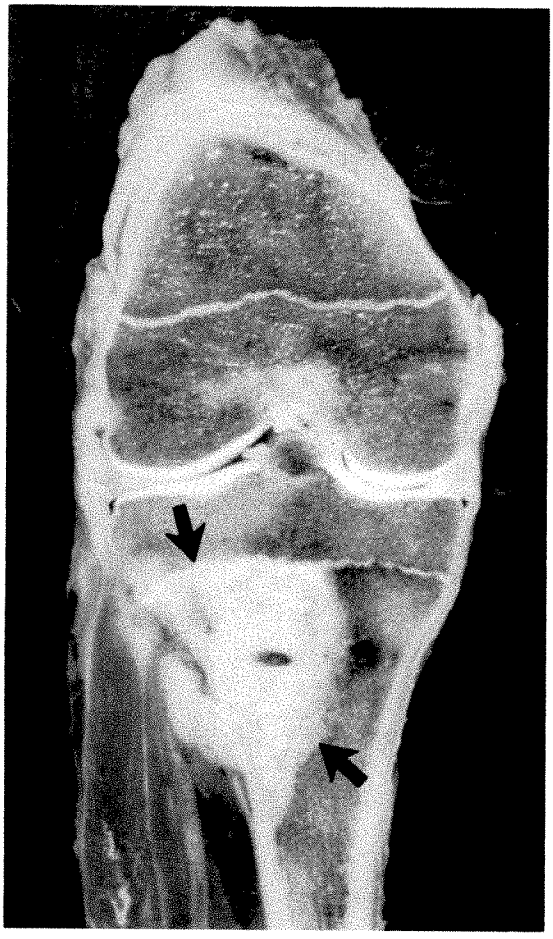


FIG. 5. Same patient as in Figure 4. Coronal section through amputated right leg shows extent of osteogenic sarcoma (arrows). Note close correspondence with strontium scans.

ence of added stable carrier strontium in the Sr^{85} solution. These possibilities are currently under investigation.

Measurement of urinary excretion of Sr^{87m} in 3 patients (Table II) indicates that virtually none of the isotope is eliminated from the body during the scanning process. This results in a high body background and is a major disadvantage to scanning with Sr^{87m}. However, since relatively large scanning doses can be given, background can be effectively eliminated by judicious use of the background cut-off circuit and appropriate contrast enhancement so that satisfactory scans can be obtained. In contrast, up to 50 per cent of the Sr^{85} dose may be eliminated via gastrointestinal and renal pathways during the 24–72 hours which elapse between injection of this isotope and the scanning procedure. In Sr^{85} scanning, this is of major significance because of the low count rate over tumor sites.[4]

The radiation dose from 100 μc of injected Sr^{87m} has been calculated to produce 2.4 mrads to the body as a whole (assuming uniform distribution) and 9.9 mrads to bone (assuming 100 per cent retention and a geometry factor of 41.6; Table I). This is to be compared with a whole body dose of 680 mrads and bone dose of 1,590 mrads from an equivalent quantity of Sr^{85}. A greater bone dose would result from the use of a geometry factor of 125 in the calculations, as was apparently done by Fleming *et al.*,[7] but this assumes homogeneous concentration throughout the body. A more realistic approach would place the actual bone dose between these two extremes.

The disadvantages of Sr^{87m} (other than the relatively high body background) are: (1) the short half-life of the Y^{87} parent—80 hours; (2) the cost of the generator, which must be renewed every 2–3 weeks; and (3) the time-consuming milking-sterilization-counting process in comparison with the ease of handling a multiple-dose radiopharmaceutical preparation such as Sr^{85} nitrate.

The results of the present investigation provide a basis for exploring additional avenues of approach to the following diagnostic problems not amenable to solution with Sr^{85}: (1) nonmalignant disorders of bone; (2) osteogenic response to hormonal, chemical, or radiation therapy of benign and malignant diseases of bone; and (3) bone disorders of children. These problems are currently under study in our laboratory. In addition, the higher doses of Sr^{87m} which are possible improve counting statistics, and thereby sharpen resolution and permit shorter scanning times in comparison with Sr^{85}. Preparation of the bowel for pelvis scans is not necessary with Sr^{87m}, as it is with Sr^{85}.[4]

SUMMARY

Sixteen patients were injected with Sr^{87m} and photoscans made of bone. Seven of these patients had metastatic cancer to bone and 4 had primary bone tumors. Except for 2 patients with eosinophilic granuloma, the Sr^{87m} scans corresponded with the roentgenograms, and in 6 patients revealed more extensive disease than did the roentgenograms. Bone biopsy in 5 patients confirmed the scan findings. In 6 patients injected with both Sr^{87m} and Sr^{85}, identical scans were obtained.

Sr^{87m} can be employed to detect metastatic cancer to bone even prior to observable roentgenographic changes. It should prove of particular value in evaluating nonmalignant disorders of bone including those of children, and in following the osteogenic response to therapy of bone diseases.

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THE SKELETAL UPTAKE OF RADIOPHOSPHORUS (P^{32})*

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IN THE course of using radiophosphorus for the treatment of patients with a diagnosis of polycythemia vera, a delayed response in one of our patients made it desirable to determine the extent to which the isotope was absorbed by the skeletal system as a whole.

A review of the literature revealed no previous study of this exact nature, but furnished a guide through animal data and autopsy studies on cases of leukemia. The average retention of P^{32} by normal mice as reported by Lawrence *et al.*[7] and Erf *et al.*[3] was 29.7 per cent and that of leukemic mice 35 per cent. Friedell and Storaasli[4] reported 33 per cent of the total injected dose remaining in the skeleton of albino rats at 14 days. Further calculations on the data of Warren[12] in humans indicate a retention of 31 per cent in chronic myelogenous leukemia at 14 days, and on the data of Bauer *et al.*[1] an average uptake of 37.2 per cent in young normal children. In the latter instance, administration was by intramuscular injection.

With a view to *in vivo* determination of the bone uptake in adult humans, we have made comparative measurements over the forehead area of our patients and over an analogous area on a phantom skull which was prepared with a known quantity of P^{32}. The forehead was selected for measurement because it offered a relatively extensive and easily accessible bony area free of large muscle masses and adjacent to brain which takes up relatively small amounts of P^{32} at the time periods measured.[6] The assumption was made that distribution in the skull bones would be uniform and that deposition of P^{32} therein would be representative of deposition throughout the skeleton since the work of Jowsey[5] has shown that the bones of adult dogs show a generally uniform distribution of adsorbed radionuclide, and Marshak[9] has stated that differences between epiphyseal and diaphyseal concentration even out with time.



FIG. 1. Phantom head constructed of balsa wood and covered with tricalcium phosphate-gelatin layer. Three of four small balsa wood spacers can be seen on the forehead area which were used to position the collimator.

MATERIALS AND METHODS

A phantom head was constructed from balsa wood (ochroma lagopus) to which was molded a mixture of tricalcium phosphate and 5 per cent gelatin (37 per cent: 63 per cent) as shown in Figure 1 to represent skull bone in both inorganic and organic composition. The average Z number of bone was taken into consideration and explains the percentages of minerals, gelatin and water used in the phantom in order to establish the same quality of Bremsstrahlung from

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FIG. 2. Oblique anteroposterior roentgenogram of phantom as "seen" by counter probe showing uniformity in gelatinous layer. The marker is 2 cm. long.

P^{32} in the phantom to the P^{32} in the bone of patients.[13] As the gel was prepared 40.3 μc of P^{32} were thoroughly mixed with it so as to obtain uniform distribution. The thickness of the calcium phosphate-gelatin skull cap was 5 mm.; this was selected as an average frontal bone thickness from roentgenograms of our patients. An oblique anteroposterior and a lateral roentgenogram of the phantom skull are shown in Figures 2 and 3. The marker is 2 cm. long. Counting over the phantom was done with a pulse height analyzer-scaler unit, adjusted to obtain the highest count rate from the P^{32} Bremsstrahlung. The cylindrical sodium iodide crystal, 3 inches in diameter $\times 1\frac{1}{4}$ inches thick was collimated with $1\frac{1}{2}$ inches of lead and recessed $1\frac{1}{2}$ inches from the opening. The end of the collimator was positioned 2 mm. from the surface of the phantom skull. It was determined that even with heavy collimation activity from some contiguous areas as well as the area immediately beneath the collimator would contribute to the observed count rate, although activity in the occipital area contributed only 1–2 per cent.

Fourteen similar studies were done on 7 patients who had received P^{32} in millicurie amounts. Lateral skull roentgenograms were taken to determine proper positioning of the counter above the frontal sinuses and also to measure frontal bone thickness. Inasmuch as skull thickness varies with heredity, age and individual characteristics of the disease, appropriate correction factors were devised and applied to relate the count rate observed over the patient to that observed over the phantom. All counts were corrected to zero time and calculations made according to the following formula:

$$\frac{A \times Pc \times 100}{Ph\ wt \times \dfrac{Ph\ c}{\mu c \text{ in } Ph} \times \mu c \text{ dose to } P} = \% \text{ in skeleton}$$

where A = estimate of weight of patient's skeleton in grams; Pc = count over patient's skull; Ph = phantom; $Ph\ wt$ = phantom skull cap weight in grams; and $Ph\ c$ = phantom count.

The estimated weight of the patient's skeleton is taken from tabulated data on the mass and effective radius of organs of the adult human body.[11] The first two terms in the denominator are known constants and may be replaced by a single numerical value, thus:

$$390.3 \text{ g.} \times \frac{9945.6 \text{ c/m}}{40.3\ \mu c} = 96{,}273.4 \frac{\text{g.} - \text{c/m}}{\mu c}$$

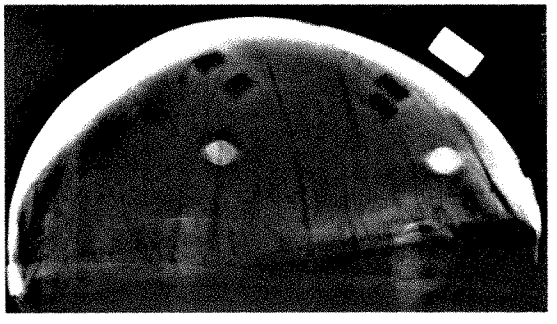


FIG. 3. Lateral roentgenogram of phantom. The marker is positioned over the forehead area where the measurements were made.

TABLE I

RESULTS OF STUDIES AFTER INTRAVENOUSLY ADMINISTERED RADIOPHOSPHORUS

Patient	History No.	Age (yr.)	Weight (kg.)	Dose (mc)	Day Measured	Per Cent Uptake
W.P.M.	-A321	73	69.5	4.9	10	36.6
W.P.M.	-A321	75	69.5	3.6	32	32.9
T.T.	-A3935	56	89.1	6.4	9	33.2
T.T.	-A3935	57	89.1	7.0	14	35.6
P.G.	-A6082	52	69.2	4.8	14	25.3
Average						32.7

This figure applies to 5 mm. thick frontal bone. When the frontal bone is 8 mm. thick, this figure becomes

$$115{,}122 \frac{\text{g.} - \text{c/m}}{\mu\text{c}}.$$

RESULTS

Data obtained from 14 patient studies are presented in Tables I and II. Measurements usually began within 24 hours after zero time and continued throughout the patients' hospital stay. In early time periods, *i.e.*, from a few hours to 3–4 days, we noted an increase of P^{32} which has been shown to be an equilibration period.[4,8,9] Thereafter, an approximate but steady ratio is established between bone and soft tissues as also demonstrated by the work of Palmer[10] and Wyard.[14] The data tabulated were therefore selected with regard to these findings except where discharge from the hospital required us to forego further measurements.

DISCUSSION

The results of the skeletal uptake of P^{32} as calculated in these studies are in the same general range as those reported in or calculated from data presented in the literature. In some cases they are a little higher than the results calculated from autopsy data, but this is generally to be expected since malnutrition and the wasting aspects of leukemia result in loss of protein, calcium, and phosphorus. However, the agreement with the animal data is good. Similar equilibration periods were observed. The general physical condition was equivalent. Our patients were of normal weight and good appetite and in no acute distress. Successive retreatment was generally occasioned by complaints of dizziness or a feeling of fullness in the head or the presence of an increasing hemogram. The object in view was to maintain the red blood cell count in the area of 6–7,000,000 cells per

TABLE II

RESULTS OF STUDIES AFTER ORALLY ADMINISTERED RADIOPHOSPHORUS

Patient	History No.	Age (yr.)	Weight (kg.)	Dose (mc)	Day Measured	Per Cent Uptake
E.B.	-A8870	72	63.4	3.5	7	14.6
W.H.	-A5587	66	94.0	4.0	7	12.2
T.T.	-A3935	58	86.1	6.0	8	23.1
T.T.	-A3935	58	86.8	6.0	14	23.9
G.B.	-A7683	76	58.9	6.0	12	20.1
W.P.M.	-A321	75	65.0	6.0	11	27.0
W.P.M.	-A321	75	62.5	6.0	14	23.8
W.P.M.	-A321	75	62.5	7.0	14	26.1
G.E.	-A11901	72	74.5	5.0	14	13.9
Average						20.5

cu. mm. and the packed cell volume below 60 per cent.

The average uptake from the intravenous route was found to be 32.7 per cent while that from the oral route was 20.5 per cent. Erf and Lawrence[2] found a similar disparity. These figures indicate the need of giving up to 50 per cent more P^{32} orally than would be administered intravenously for equivalent therapy. Some patients in our series had either all intravenous or all oral doses. However, 2 of our patients (W.P.M. and T.T.) have had a long series of both intravenous and oral doses. Data concerning the amounts administered and intervals before readministration was required in these patients indicate the relative effectiveness of these two modes of administration. The average of all intravenous doses for both patients was 5.2 mc administered every $8\frac{1}{2}$ months. The average of all oral doses was 6.5 mc administered every $3\frac{1}{2}$ months. From the former, 1.58 mc of P^{32} would have been taken up and from the latter, 1.33 mc. It seems apparent in these cases that an average oral dose which was only 25 per cent greater than the average intravenous dose was not equivalent in maintaining suppressed marrow activity, hence the shortened interval between successive administrations.

The measurements were made *in vivo;* they are easily performed with standard radioisotope laboratory equipment and are serving as a check on the patients' progress by integration with clinical state, size and frequency of doses, as well as the other routinely used hematologic tests.

SUMMARY

Comparative measurements have been made on a phantom skull and on patients under treatment for polycythemia vera using P^{32}. The uptake of P^{32} by the skeleton has been calculated.

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DISTURBANCE IN BONE GROWTH PRODUCED BY 3.5 MEV. ELECTRON IRRADIATION OF YOUNG RABBIT LIMBS*

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IT IS well recognized that irradiation of the growth centers of bone can lead to inhibition of growth, or deformity, or both.[5,8] This is a hazard of radiation therapy of children, especially where malignant soft tissue lesions near the epiphyses are targets of curative roentgen ray doses. Methods to reduce or eliminate the complications of such therapy have been sought for many years. The ideal would be to deliver an effective dose to a tumor while completely sparing the bone. The use of high energy particulate radiation, sources of which are becoming available, would seem to offer a promising approach because of the favorable pattern of energy release within tissues. With this form of therapy there is a rapid build-up of released energy beneath the surface to a peak level with a relatively sharp drop as one goes deeper. In the case of a superficial tumor at a distance from underlying bone, it should be relatively easy by adjusting the voltage to deliver through a single portal high doses of electrons to the lesion while sparing the bone. However, when the tissue to be treated is contiguous to normal bone, the "tail" of the energy distribution may include enough of the epiphysis to cause damage (Fig. 1). The resulting asymmetric disturbance in bone growth might prove more difficult to correct than a more uniform shortening, which is to be expected after high energy x-irradiation through a similar portal.

In the present report an experiment is described in which a comparison was made

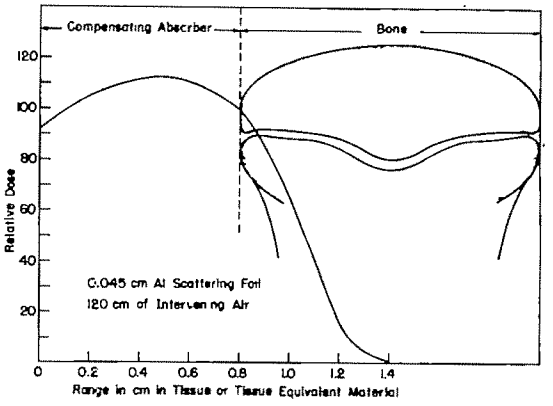


FIG. 1. Distribution of depth dose for 3.5 mev. electrons with cone as used in this experiment. A schematic line drawing of the proximal tibia is superimposed to demonstrate the rapidity of the fall-off through bone. An average bone density of 1.65 gm./cc. has been assumed and used for all calculations.

of the effects of high energy x-ray versus electron irradiation on the growth of bones of limbs of young rabbits to ascertain whether the inhibitions or deformities of growth would follow the patterns predicted.

MATERIALS AND METHODS

Forty male albino rabbits, $5\frac{1}{2}$ to $6\frac{1}{2}$ weeks of age, were randomly divided into 2 groups of 20 animals each. The first group received x-ray and the second group electron irradiation. The average weight of the animals in Group I was 3 lb., 1 oz.; in Group II, 3.0 lb. The animals were anesthetized, using intravenous veterinary nembutal and positioned with the right side down. The right hindleg was then extended fully with traction ex-

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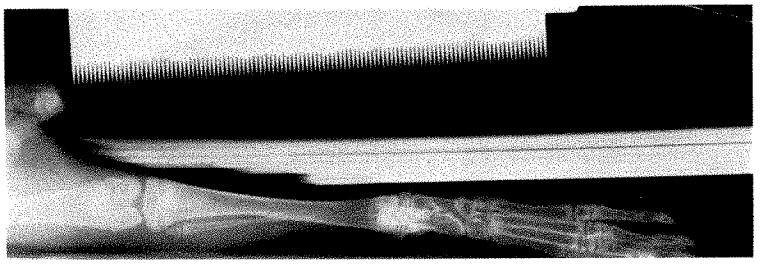


Fig. 2. Sample roentgenogram obtained at time of electron treatment. Compensating absorbers have been introduced into the beam to equalize the dose along the medial aspect of the irradiated soft tissues adjacent to bone. The proximal limit of the treatment field is identified by the end of the serrated marker and extends well above the upper tibial epiphysis.

erted by a tension bandage secured to the right ankle and was irradiated to include all of the tibia and the foot through a medial entry portal. The body, tail, and left hindleg were excluded from the beam.

Group I was treated with x-rays generated at 2 mev. by a Van de Graaff machine. The target to skin distance was 71 cm. Sufficient unit density absorber (masonite) was interposed so that the maximum dose was at the level of the skin surface. Masonite was also placed below the hindleg to provide electron equilibrium back scatter. The maximum dose was estimated to be 1,000 rads and the minimum dose, 950 rads at the exit side.

In 19 of the 20 animals in Group II, the right hindleg was irradiated in similar fashion but 3.5 mev. electrons from a Van de Graaff accelerator were used. Compensating absorbers, equivalent to 0.1 to 0.7 cm. of tissue, were introduced into the beam in order to equalize the dose in the treated tissues adjacent to the bones on the medial side (Fig. 2). The estimated dose in soft tissue immediately adjacent to bone was 1,000 rads and fell rapidly to lower values beyond this region (Fig. 1). Proper positioning of the absorbers in relation to the irradiated part was checked for each rabbit by roentgenograms prior to treatment (Fig. 2). The twentieth animal was inadvertently given 2,000 rads.

For the purposes of this study we adopted 1.65 gm./cc., as proposed by Haas and Sandberg[6] after studies on the calvarium and mandible, as the average density for bone. This value seemed appropriate in view of the immaturity and small size of the tibias in young rabbits and was selected in preference to 1.85 gm./cc., as suggested by Spiers,[11] based on studies of compact femoral bone.

The animals were followed for 6 months. They were examined clinically for evidence of cutaneous radiation reactions, epilation, deformity, and growth retardation at least at monthly intervals. Weights were recorded at the same time.

Roentgenograms of both hindlegs in extension with the animals prone were obtained before treatment and each month thereafter for 4 months. The lengths of the right and left tibias of each animal were measured on roentgenograms, using standard reference points.

Measurements were also made on the left side, which was used as a control. Baserga *et al.*[2] were unable to find any difference in the lengths between the right and left tibias in unirradiated rats. Regen and Wilkins[9] have shown that in rabbits there was no difference in the lengths of the radii and ulnae in unirradiated controls and those of the untreated foreleg in treated 25 day old animals.

A radiopaque ruler was included for each set of roentgenograms and the measured lengths for all bones were reduced by an appropriate factor to correct for magnification. The animals were sacrificed 6 months after treatment, and the hindlegs removed. Both limbs were dissected free of soft tissues and the actual lengths of the tibias

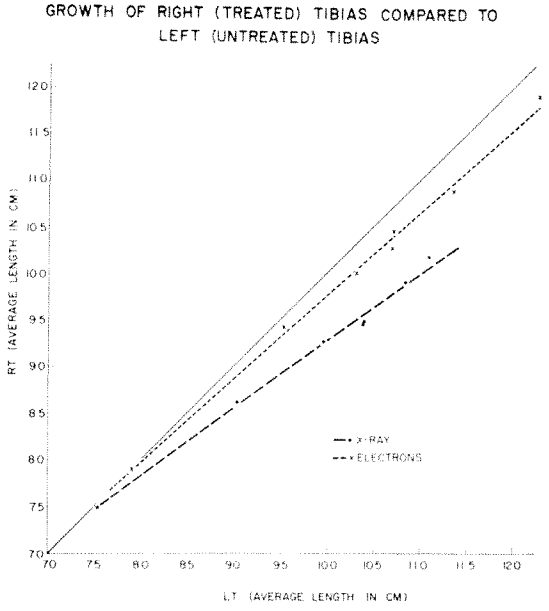


FIG. 3. Lengths of the tibias in all animals of Groups I and II were averaged at each time interval and the values are shown on the scatter diagram. The best-fitting straight lines were calculated for each of the two groups. They can be compared with each other and with the 45° plot (solid line) which indicates the slope for equal growth of the right versus the left tibia. There is greater shortening of the x-irradiated tibias than of those receiving electron irradiation. The slopes of both lines differ significantly ($p < 0.01$) from the 45° plot.

were measured, using the same points of reference. Seventeen animals in Group I and 15 in Group II were considered suitable for detailed study. The 7 omitted died or sustained leg or spine fractures, and were not included in any of the calculations.

RESULTS

The measured lengths of the tibias of each animal for each month investigated were used to compute regression lines of right on left. The individual points fell very close to a straight line and, in fact, the values of r (the correlation coefficient) were in excess of 0.9 for all animals but one. The slopes of these lines were calculated and were averaged for the x-ray group and for the electron treated group. These average slopes than were tested for significance, using Student's t. The average slope of the 17 x-irradiated rabbits was 0.718 and the standard deviation, 0.114. For the 15 animals in the electron group, these values were 0.087 and 0.094, respectively. This analysis shows a significantly greater reduction in tibial growth rate (right versus left) in the x-irradiated bones than in the electron-irradiated ($p < 0.001$).

The analysis of the data given above was performed by computing individual regression lines for each rabbit and then averaging the slope coefficients. For clarity of visual presentation, the regression lines fitted to the average lengths measured each month are shown in Figure 3. The two methods gave results which were in close agreement.

Foot shortening was not measured but the pattern corresponded to that of the tibias. The muscle mass of the right hindleg in Group I animals was reduced (Fig. 4). Group II rabbits showed no appreciable change in this regard.

Genu varum developed in both groups, of slight degree in Group I, but more obvious in Group II, where 3 of 15 animals had 10 to 15 degree deformities (Fig. 5). These clinical observations were confirmed by measurements made on the roentgenograms. The change was coupled, in some Group II rabbits, with an internal rotational deformity of as much as 25 degrees. These alterations induced in the electron irradiated group were seen in exaggerated form in the rabbit given 2,000 rads, which developed pronounced genu varum deformity with marked (70 degree) internal rotation at the knee. Group II animals showed an additional growth disturbance in that the normal sinuous curve of the rabbit tibia was straightened, thus accentuating the genu varum (Fig. 5).

The animals in both groups lost hair along the medial aspect of the irradiated limb. The epilation, never pronounced, reached its maximum in the second month and was more conspicuous in Group II than in Group I. Complete regrowth of hair was evident in all animals by the fourth month. Erythema and other evidences of radiation

reaction were of no consequence at any time.

The growth of the rabbits proceeded equally in Groups I and II. The average weight at the time of sacrifice for each of the two groups was 8 lb., 5 oz.

DISCUSSION

The inhibition of bone growth by the x-irradiation of epiphyses has been well documented, experimentally and clinically. In rabbits, this effect was demonstrated many years ago by Brooks and Hillstrom,[4] while Barr *et al.*[1] showed similar results in dogs. Bisgard and Hunt[3] have shown in rabbits similar in age to those of the present experiments that single exposures of 200 r can cause histologic changes in the epiphyseal cartilage. A dose of 400 r to the entire extremity, however, was the minimal dose required to cause grossly measurable inhibition of bone growth. Hinkel[7] showed in rats that a 500 r exposure of an area 5 mm. in diameter at the knee was the minimal dose which caused measurable growth retardation.

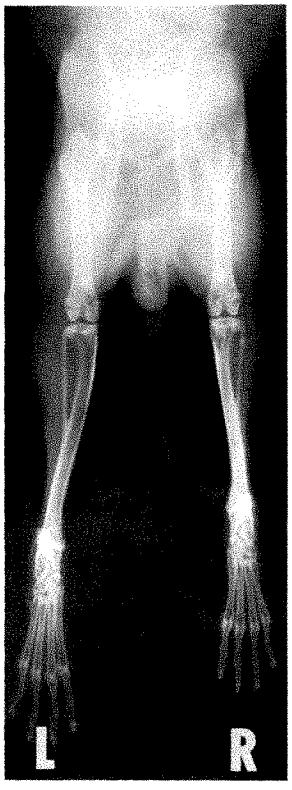


FIG. 4. Roentgenogram of x-irradiated rabbit made 6 months after delivery of 1,000 rads to the soft tissues of the right hindleg. There is shortening of the right lower hindleg and foot and diminution of muscle mass as compared to the left side. Minimal genu varum deformity also is present, as well as less sinuosity of the tibial shaft. The genu varum and sinuosity of the right (irradiated) tibia are similar to those of the younger animal of Figure 2. The unirradiated tibia shows the increase in sinuosity and genu valgum due to age.



FIG. 5. Roentgenogram of postmortem dissected specimens of the hindlegs of electron irradiated rabbit 6 months after delivery of 1,000 rads medially to the soft tissues of the right hindleg. A comparison with the unirradiated left side shows pronounced genu varum deformity and straightening of the normal sinuous tibial curve. There is also some shortening of the right hindleg.

In the cited experiments bone shortening as a result of x-irradiation was noted by all, but no deformities were mentioned except by Hinkel,[7] who found bowing in 2 rats. These animals received doses of 1,000 and 1,500 r, respectively, delivered through a medial portal. He ascribed the bowing

either to a direct effect on the epiphysis or to "changes secondary to modified muscle stresses and mechanical imbalance caused by the short leg, *per se*." No reports were found in the literature concerning the growth disturbance produced by electron irradiation.

In the presently reported series the asymmetry of inhibition of bone growth caused by electron irradiation resulted in appreciable deformity, although it did not influence greatly the growth in length of the exposed bones. The portion adjacent to the soft tissue which received peak dosage was most affected, while that on the opposite side was virtually unaffected. The genu varum deformity, while not severe in rabbits receiving 1,000 rads, was pronounced in the single animal receiving 2,000 rads. With the higher dosage required for the treatment of malignant diseases in children, undoubtedly even more severe deformities would result. With the use of a single portal, the effects on the one side of the epiphysis might well lead to marked inhibition of bone growth, while the other side of the epiphyseal cartilage would receive only Bremsstrahlung and thus remain virtually unirradiated.

X-irradiation of the growing epiphysis produced mainly a uniform shortening with very little genu varum deformity. Bowing similar in degree to that described by Hinkel[7] in rat femurs was not seen in any of our animals. We believe that the minimal change we noted in the tibial curvature was the result of two factors: the slight difference (5 per cent) in dose distribution across the epiphyseal plate and the underdevelopment of the x-irradiated bone (Fig. 4 and 5).

The muscle mass on the x-irradiated side was found to be reduced in Group I rabbits. This could have been the result of disuse and underdevelopment occasioned by the bone shortening, but the reduction seemed out of proportion to the degree of shortening. We believe this to be the result of direct x-ray effects on soft tissues, and will report more on this subject in another communication. The study by Rubin and his colleagues[10] supports this view. They irradiated the epiphyses, the diaphyses or the metaphyses of the tibias and femurs in growing rats, and compared the effects with those produced by irradiation of the entire bone. While their study was concerned with the aberrations of skeletal development which resulted, it is noteworthy that in their illustrations the reduction in muscle mass appeared to be independent of shortening. The governing factor would appear to be the amount of muscle, not bone, which was irradiated. Asymmetric soft tissue changes may also have had a causative part in this deformity and in the loss of the normal sinuous curve noted in the Group II animals.

The main objective of our experiment was to determine the effects of electron irradiation of soft tissues contiguous to growing bone where exposure of the bone is an accompaniment of the therapy. These were compared to those produced by x-irradiation where an identical dose was delivered to the soft tissues adjacent to bone, using a similar treatment portal. The results show that pronounced abnormalities can be occasioned by either form of treatment. Since correction of a uniformly shortened bone is less complicated than that of a bowed or otherwise deformed limb, orthopedic considerations should be prominent among the factors weighed when radiation therapy modalities are being considered for children.

SUMMARY

The effects of high energy x-irradiation and electron irradiation on the growing knees of young rabbits were compared experimentally. The principal abnormality in those animals receiving x-irradiation was shortening of the treated limbs, while in those irradiated with electrons, it was genu varum.

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ADDENDUM

Since the submission of this manuscript, D'ANGIO, G. J., GOTTSCHALK, A., and LYMAN, J. T. (unpublished observations) have established the density of 4 week old rabbit distal femurs including the epiphyses as 1.22 gm./cc. This results is no important corrections in the shape of the curve shown in Figure 1.

We wish to acknowledge with gratitude the assistance of the Misses Yvonne Bishop and Barbara L. Brown for preparing the statistical portions of this paper.

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A FLUOROSCOPICALLY CONTROLLED ADJUSTABLE MIDLINE LOCALIZER FOR STEREOTAXIC SURGERY*

A MODIFICATION OF THE GUIOT-GILLINGHAM TECHNIQUE

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UNTIL it was shown by Meyers[12] that surgical interruption of the pallidofugal fibers could modify the rigidity in the treatment of Parkinsonism, surgical treatment of dyskinesias had been largely confined to interruption of corticospinal pathways, the results of which were hazardous and not encouraging. The introduction of the stereotaxic apparatus in 1947 by Spiegel *et al.*[15] eliminated the formidable and more hazardous open operative approach. Since that time, many other stereotaxic techniques have been developed.[1,2,10,13,14,17,18] Most of these procedures require a stereotaxic pneumoencephalogram to relate midline structures to external markers. Also, the treatment probe is usually put into place as a separate stage at a later time.

Many attempts have been made to simplify the stereotaxic technique of treating dyskinesias. Unfortunately, attempts at simplification frequently are accompanied by loss of accuracy in the placement of the treatment probe. On the other hand, those methods where accuracy is preserved are usually attended by a series of procedures entailing prolonged and tedious maneuvers using complex and cumbersome apparatus.

The Guiot method,[8] as modified and applied by Gillingham,[7] appears to be among the simplest of stereotaxic procedures which preserve accuracy.* However, it has been found by us to be a relatively time consuming procedure in which the lesion cannot be made closer than 15 mm. to the midline. It is often preferable to make the lesions as close as 11 mm. to the midline.[3]

In an attempt to make stereotaxic treatment of dyskinesias a simpler, shorter and more flexible procedure without sacrificing accuracy, we have modified this technique. The surgical preparation and placement of the therapeutic probe is now carried out in one stage, allowing the lesion to be made virtually anywhere within the brain, as close as 1 cm. to the midline and without loss of accuracy.

METHOD

The patient is first taken to the operating room where the following procedure is carried out under local anesthesia:

1. The approximate midline is determined by placing a silk thread from the glabella to the inion over the scalp in the sagittal plane. Through small scalp incisions the specially designed base plates for the stereotaxic instrument (Fig. 1, *A* and *B*) are screwed to the skull in the midline; one in the frontal region, one at the vertex and one in the occipital region. It is important that the long axes of the base plates be as perpendicular to the sagittal plane of the skull as is possible. A graduated sliding bar (Fig. 1*A*) is then inserted into each of the base plates. Attached to each bar is a turret with (a) a hole to receive a wire, and (b) a ball and socket joint to

* The Guiot-Gillingham stereotaxic apparatus which was modified is available from Allen and Hanbury Co. Ltd., London, England.

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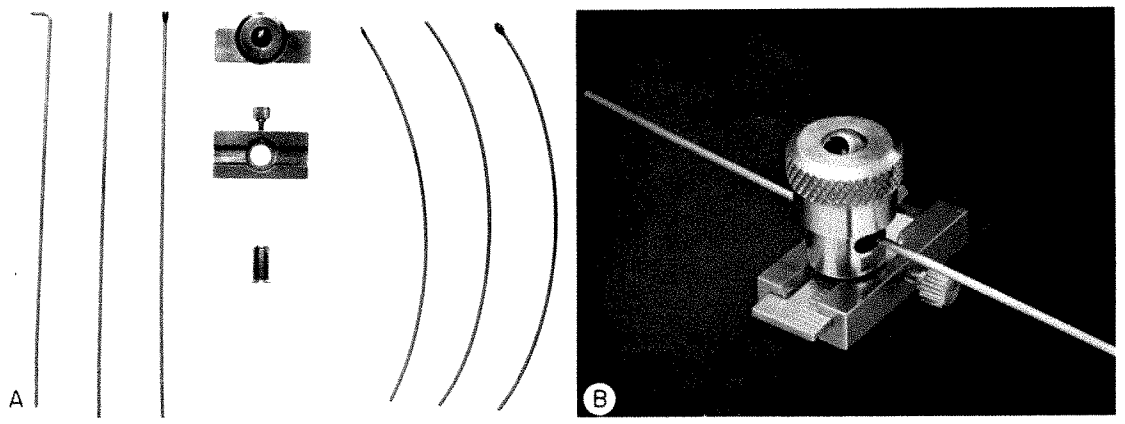


Fig. 1. Disassembled adjustable midline localizer showing screw, base plate and sliding bar with ball and socket joint. Also shown are the straight and curved wires for insertion into turret of sliding bar. (*B*) Assembled adjustable midline localizer.

receive the midline Guiot frame. A separate length of wire (Fig. 1*A*) is then inserted into the holes in each of the three turrets and secured. The anterior end of the frontal wire is bent at a right angle, and a metallic ball is attached to the posterior end of the occipital wire so that during fluoroscopy, the wires can be identified. The wires are made of .054 gauge tempered stainless steel piano wire and are not malleable. Curved and straight wires were made to conform to varying configurations of the skull. Figure 2*A* shows the adjustable midline localizer attached to a patient.

2. A parasagittal frontal burr hole is made and a ventricular catheter is inserted, through which contrast material and air will be introduced into the lateral ventricles (Fig. 2, *A* and *B*).

3. A posterior parietal incision and a 17 mm. burr hole, for subsequent insertion of the treatment probe, is made on the side which is to be treated. It has been found that the approximate location of this burr

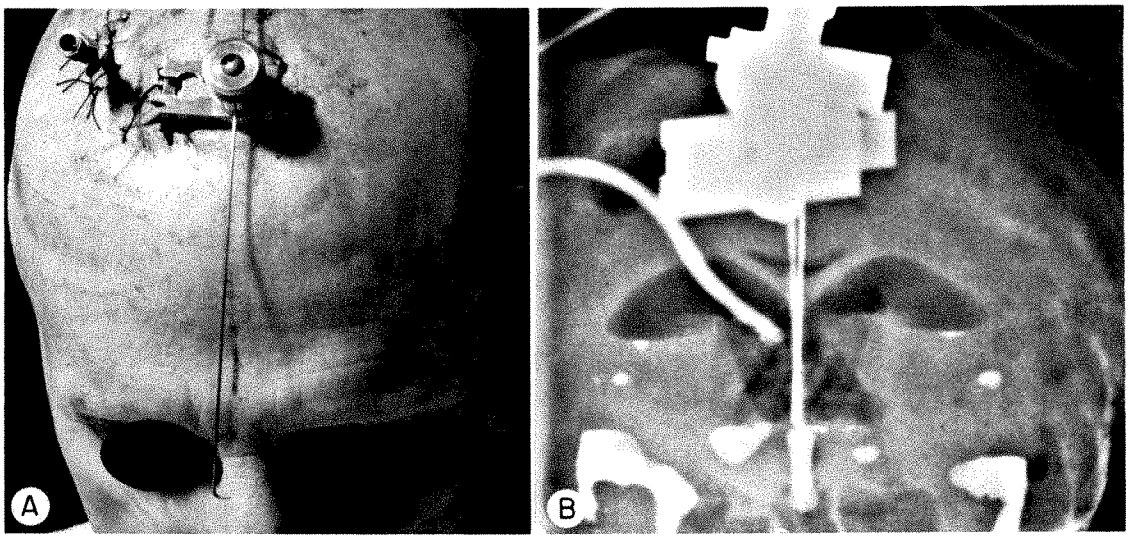


Fig. 2. (*A*) Adjustable midline localizer attached to skull of patient. Note the frontal ventriculostomy tube in place. (*B*) Frontal fluoroscopic roentgenogram made during the process of adjusting the wires of the localizers to the midline.

hole should be 8 to 9 cm. above the external occipital protuberance and 1.5 cm. off the midline. In order to allow more latitude for the introduction of the probe, the anterior and posterior lips of the burr hole are removed with bone forceps approximately 5 mm. in each direction. If the external occipital protuberance cannot be palpated, the location can be determined from the plain skull roentgenograms. While it is frequently advisable to have made a routine pneumoencephalogram as a screening procedure, it is not necessary for the modified method of stereotaxis being described. The wounds are closed in one layer and a light dressing is applied.

The patient is then moved from the operating table to a chair adapted to our roentgen image intensification equipment. The chair also allows for the attachment of a special head holder for patients with violent head movements. With the patient's head flexed, 20 cc. of air is exchanged with cerebrospinal fluid through the catheter in the frontal burr hole. If the head is extended, and then quickly flexed, air is frequently trapped in the posterior part of the third ventricle, which outlines the posterior commissure (Fig. 3 *A*, P.C.). Three cubic centimeters of pantopaque are then introduced through the same catheter with the head flexed. With the head inclined slightly to the side opposite the ventricle containing the pantopaque, the head is slowly extended while the patient is viewed fluoroscopically in the lateral projection. This maneuver will generally fill the third ventricle with pantopaque, outlining the anterior commissure (Fig. 3*A*, A.C.). If the air failed to outline the posterior commissure in the first maneuver, further extension of the head will generally outline the posterior commissure as the pantopaque reaches the aqueduct (Fig. 3*B*). Polaroid and conventional lateral fluoroscopic spot roentgenograms are taken to record these landmarks. The patient is then put into the frontal projection and the head is rotated until the third ventricle, which is outlined with pantopaque, and the septum pellucidum, which is outlined with air, are viewed perpendicular to their long axes. The wires are then adjusted under fluoroscopic control until they coincide with the long axis of the third ventricle (Fig. 2*B*). When the wires are in the midline, a set screw on each base plate is tightened (Fig. 1, *A* and *B*). This prevents the sliding bars which hold

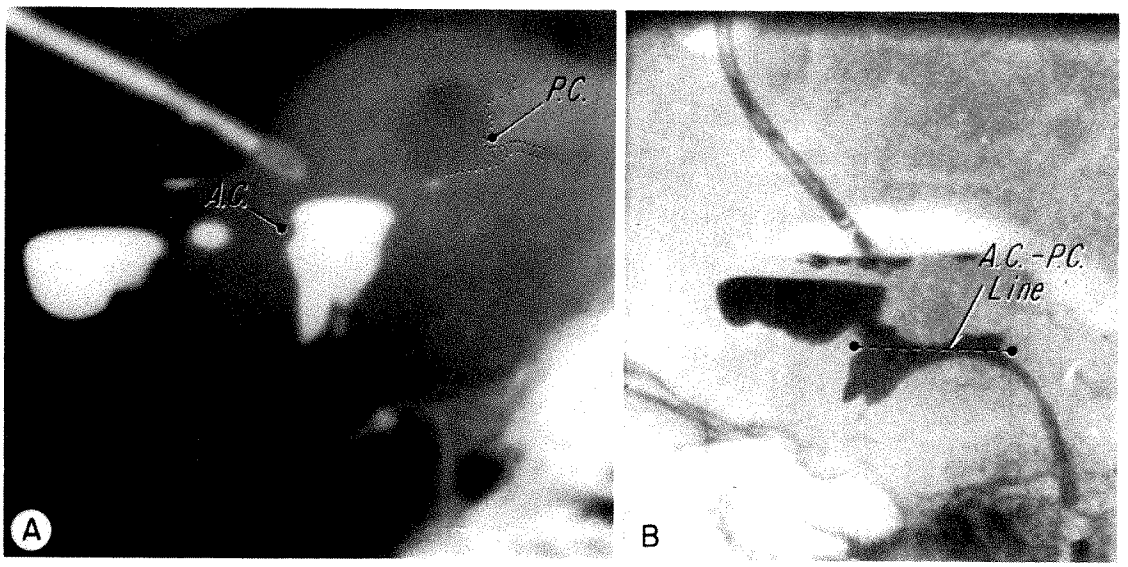


FIG. 3. (*A*) Lateral fluoroscopic roentgenogram showing the posterior commissure (P.C.) outlined with air and the anterior commissure (A.C.) outlined with pantopaque. (*B*) Lateral fluoroscopic Polaroid roentgenogram showing A.C.and P.C. outlined with pantopaque and the A.C.-P.C. line indicated.

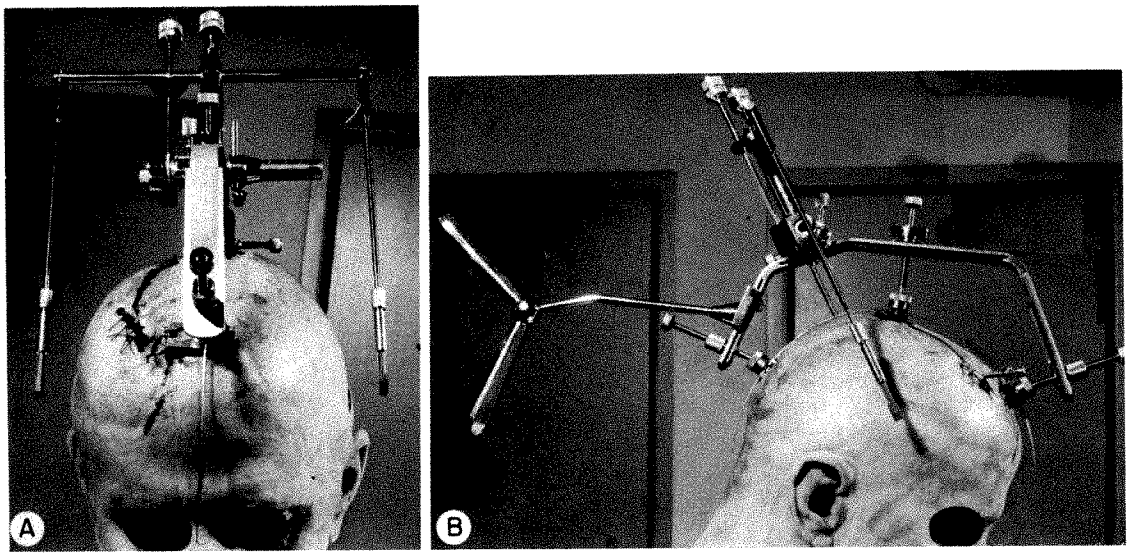


Fig. 4. (*A* and *B*) Guiot-Gillingham frame attached to adjustable midline localizers, after the midline has been established.

the wires from moving out of place. The position of each of the bars is recorded from the gauge markings on them (Fig. 1, *A* and *B*) so that the same settings can then be used to find the midline if the other side of the brain is to be treated at a later date.

The median frame of the Guiot-Gillingham apparatus is now fixed to the ball and socket joints on the sliding bars (Fig. 4, *A* and *B*). The remainder of the procedure is as described by Guiot,[8] Gillingham *et al.*,[7] and Donaldson and Gillingham.[6] While the patient is being fluoroscoped in the lateral projection, external localizers, which indicate the angle of approach of the treatment probe to the target point, are positioned (Fig. 4*B*). Fluoroscopic spot roentgenograms are made to determine the position and approach angle of the external localizers to the target point relative to the line connecting the anterior and posterior commissures (A.C.-P.C. line). When the positioning is satisfactory, the depth that the treatment probe is to be inserted to attain this position is measured from the external localizers.

The treatment probe, which in our hands is currently an indwelling depth electrode, is introduced through the line of incision over the posterior parietal burr hole and passed through the parietal lobe, the trigonal region of the lateral ventricle, the pulvinar of the thalamus, and into the target point (the region of the ventral lateral nucleus). It has been our experience that if the placement of the electrode is quite good and the patient is one who is likely to respond readily to the treatment, one frequently sees improvement in the clinical state of the patient simply with the insertion of the electrode into the thalamus. The depth electrode is attached to the skin and the apparatus is removed. Roentgenograms are made of the final position of the depth electrode (Fig. 5, *A* and *B*). The patient is returned to the operating table where the incisions are cleaned, the frontal catheter removed, and the wounds closed. During the following week, repeated sessions of stimulation and recording via the depth electrode are carried out. This determines the accuracy of the electrode placement according to physiologic criteria. A lesion is then gradually made over a period of several days with electrocoagulation. The depth electrode is not removed until the maximal therapeutic benefit is obtained, which is usually within 7 to 10 days.

In order to obtain the roentgenograms for localization in the shortest possible

time, we have constructed a special frame on the fluoroscope so that the Polaroid cassette can be dropped quickly into place for spot filming. The processing time of less than 30 seconds per film with the Polaroid technique, as compared with 7 to 10 minutes per film by conventional techniques, saves 30 to 60 minutes during the procedure. To facilitate the use of the Polaroid films in localization, a horizontally positioned view box is used and a piece of transparent celluloid is placed over each of the Polaroid films obtained. By tracing anatomic structures onto the transparent celluloid, and then placing this over each of the subsequent Polaroid films, the relative relationships of the pertinent anatomic landmarks can be determined. In order to compensate for geometric distortion, a frontal and lateral roentgenogram are taken with an opaque centimeter scale attached to the head at the plane of the target point.

DISCUSSION

The results of the use of this technique, which have been very encouraging, and the clinical details will be reported separately. In selecting the position where the lesion is to be made, current neurosurgical opinion holds that the region of the ventral lateral nucleus of the thalamus is the area of the brain which, when destroyed, leads to the most satisfactory alleviation of dyskinesias.[3,5,9,16] This area can most closely be approximated by coordinates estimated from the A.C.-P.C. line (Fig. 3*B*). We have been making our lesion just above and on the A.C.-P.C. line at the junction of the posterior and middle thirds, 11 to 15 mm. from the midline. The locations were chosen on the basis on the anatomic work of Van Buren and Maccubbin,[19] and Hassler.[9]

There is now mounting evidence of the safety of pantopaque in the cerebrospinal fluid pathways. It has been used clinically for many years, usually without sequelae in both the spinal[4] and ventricular[7] cerebral spinal fluid spaces. In addition, a recent experimental study in dogs has shown no effect on the ependyma after as long as 6 months of continuous contact with pantopaque. The arachnoidal inflammation which did occcur was transient, healed with residual fibrosis within the pia arachnoid only, and caused no changes in the subjacent brain parenchyma.[11]

SUMMARY

A fluoroscopically controlled method for the stereotaxic treatment of Parkinsonism and other dyskinesias, modifying the Guiot-Gillingham technique, is described. The procedure is performed in one relatively short and simple stage, and allows increased flexibility in the positioning of the treatment probe. This method is of

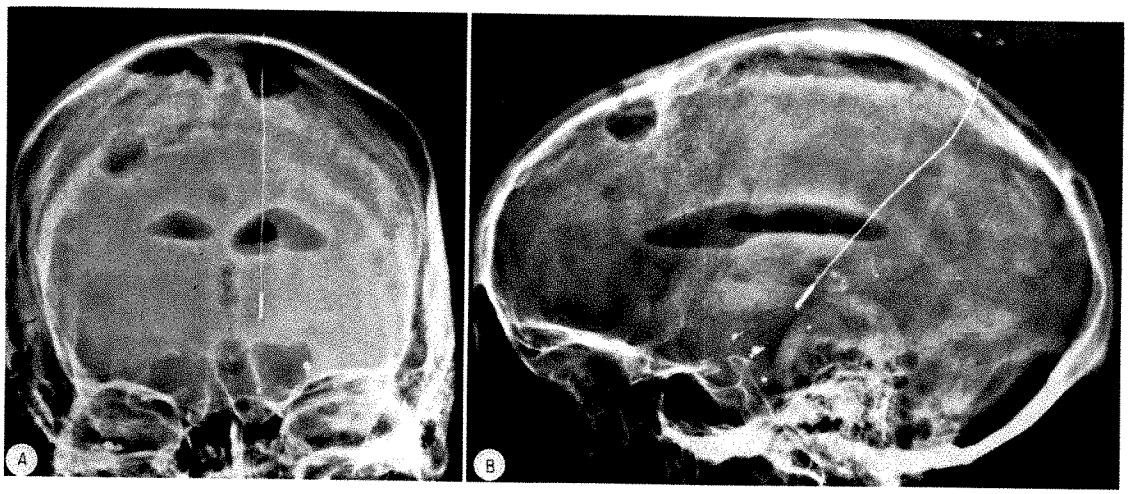


FIG. 5. (*A* and *B*) Roentgenograms made after placement of the depth electrode.

special interest to the radiologist since in it, he is an integral member of the surgical team.

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The role of Mr. Leonard Aberbach, Medical Electronic Technician, National Institutes of Health, in the design and construction of the Polaroid cassette holder for our fluoroscope is gratefully acknowledged.

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A NEW SOMERSAULTING CHAIR FOR CEREBRAL PNEUMOGRAPHY*

By D. GORDON POTTS, M.D., *and* JUAN M. TAVERAS, M.D.
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MANY different forms of support have been used for patients during ventriculography and pneumoencephalography. Some departments seat the patient backwards on an ordinary chair and then transfer him to a couch for the prone and supine roentgenograms. Robertson[5] described a chair, devised by Wilson, that is jointed so that the back can be lowered and the footpiece raised to convert it into a stretcher and Lindgren[4] described a more versatile type of convertible chair-couch for use during encephalography. A detachable support for use on an ordinary hospital stretcher during lumbar encephalography was used by Scott.[6] Taveras[7] described an axial rotation table which can be used for prone, supine, or lateral recumbency roentgenograms.

Somersaulting techniques have been used for many years in some departments for children having pneumoencephalography or ventriculography, but the mechanical problems of somersaulting a heavy patient limited its use in adults. Garcia Oller[3] devised apparatus which enabled an adult patient to be somersaulted completely in a forward or backward direction, and this provided an efficient method of manipulating the air into the different parts of the ventricular system, but unfortunately the large circular metal supports excluded the use of a standard skull roentgenographic table. Amplatz[1] used a chair in which a patient could be somersaulted or turned into the lateral recumbent position, but it also was not ideal for use with the skull roentgenographic table.

A chair was designed at the Neurological Institute, New York, which enables the patient to be somersaulted as many times as necessary in a forward or backward direction during pneumoencephalography or ventriculography. This chair may be used in conjunction with a Franklin or similar skull table. This chair has now been used for over 1,500 studies and it has proved a convenient and efficient method of manipulating air into all parts of the ventricular system or subarachnoid spaces, and it eliminates most of the heavy lifting usually associated with these studies.

DESCRIPTION OF THE CHAIR

The chair is mounted on 4 wheels and 2 of these wheels run on a metal rail which maintains the position of the chair so that it is always centrally placed in relation to the skull table Bucky grid. It is rotated around a horizontal axis by an electric motor or a handle which turns it through a reduction gear. In addition to this somersaulting motion, the chair may also be rotated around a vertical axis. The back of the chair and the footrest are both adjustable to accommodate tall or short patients, and behind the patient's lumbar region is a large defect in the chair through which a lumbar puncture may be performed. The patient's head may be immobilized by a head clamp attached to the skull unit or by a radiolucent head holder attached to the top of the back of the chair.

USE OF THE CHAIR FOR PNEUMOENCEPHALOGRAPHY

When loading the patient into the somersaulting chair, it is first brought into the erect or sitting position and the stretcher on which the patient is lying is brought in line with the chair so that the head end of the stretcher is adjacent to the front of the

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chair. The patient is then brought into the sitting position on the stretcher and moved backwards until he is sitting in the chair. The stretcher is removed and the legs allowed to flex at the knees to bring the feet onto the footrest. He is then strapped into position and the chair is wheeled up to the skull unit until the patient's forehead is resting on the Bucky grid (Fig. 1). At the Neurological Institute, New York, the head is held in position by a self-centering head-holder, one side of which forms a film holder for lateral roentgenograms and the other side is a lucite plate. The lumbar puncture is performed in this position and filling of the ventricular system and subarachnoid systems is achieved in the usual way, the degree of filling being checked by polaroid films. Posteroanterior and lateral roentgenograms are taken at every stage in the fractional technique, and if necessary, lateral autotomograms are also made. A ceiling-mounted tube is used for all of the lateral roentgenograms and the skull table is used for all anteroposterior or posteroanterior roentgenograms. When the ventricular system and subarachnoid spaces are adequately filled with air, the patient is moved away from the skull unit and turned into

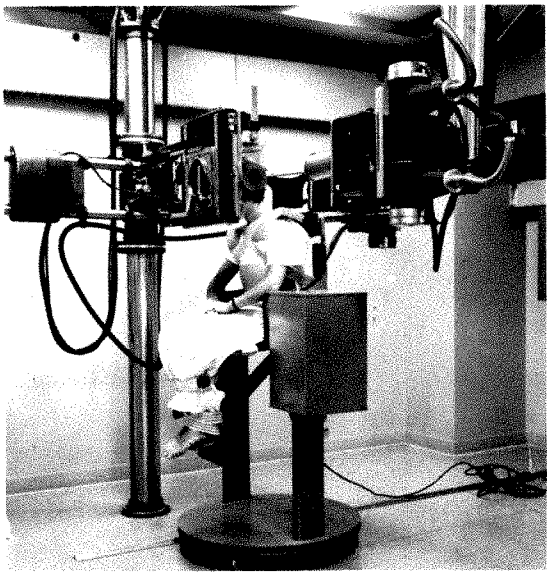


Fig. 1. Patient in sitting position during pneumoencephalography.

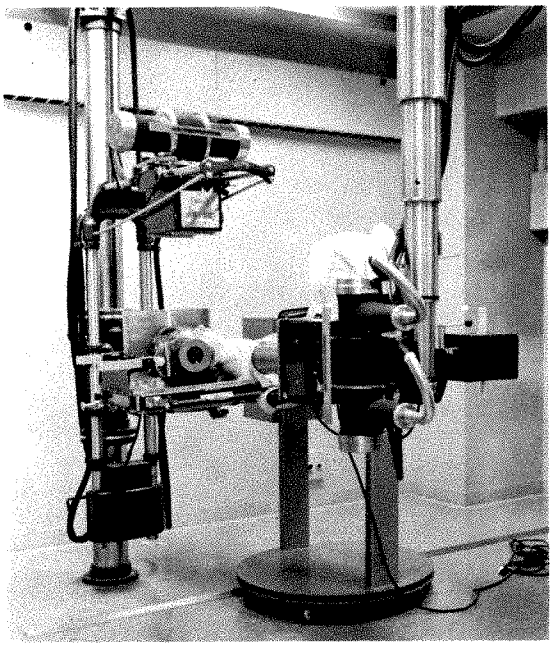


Fig. 2. Supine or "brow-up" position.

the supine or brow-up position. The chair is rotated through 180° on its vertical axis and it is then moved toward the skull unit until the patient's head is lying centrally on the Bucky grid (Fig. 2). Exposures in anteroposterior, lateral, and Towne projections are made at this stage. If the anterior end of the third ventricle is not filled in this position, the chair is tipped down a little, or the head is extended into a hanging-head position and a coned lateral roentgenogram is obtained. The patient is now brought up into the sitting position and then over into the prone or brow-down position and the chair again rotated 180° on its vertical axis to bring the head onto the skull table (Fig. 3). Posteroanterior, reverse Towne, and lateral roentgenograms are then obtained. The back of the third ventricle and fourth ventricle probably will not be visualized when the maneuver is performed in this way but these are usually seen on the roentgenograms made in the sitting position. If these parts of the ventricular system have not been clearly shown, the method described below for ventriculography should be used. In order to fill both temporal horns at the same

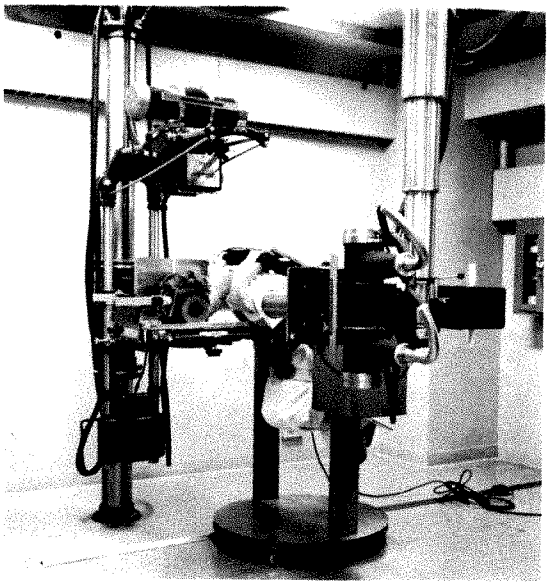


FIG. 3. Prone or "brow-down" position.

time, the patient is manipulated from a brow-down position through the upside-down position into the brow-up or supine position, that is, he performs a forward somersault. Orbital, Towne and stereolateral roentgenograms are then made. For stereolateral roentgenograms a cephalocaudad tube shift is used so as to separate the two temporal horns which are both simultaneously filled by the forward somersault (Fig. 4, *A–D*). In most cases this series of manipulations will give complete visualization of the lateral ventricles, but if very little air is present in the ventricles, the number of positions of the patient may be increased to demonstrate areas not already shown. The lumbar puncture needle may be left in position throughout all of these manipulations and, if further air is required at any stage, it may be introduced.

USE OF THE CHAIR FOR VENTRICULOGRAPHY

The patient is strapped in the chair in the same way as for pneumoencephalography and turned into the brow-up or supine position and anteroposterior, lateral and Towne roentgenograms are made. He is then moved through a complete forward somersault and brought into the supine position. This fills the temporal horns without bringing the air into the region of the foramen of Monro and therefore no air is lost during this maneuver. Orbital, Towne, and stereolateral roentgenograms are then obtained. Next, the patient is brought into a sitting position to empty the temporal horns and then manipulated through a slow backward somersault into the brow-down position. This fills the posterior part of the third ventricle, the aqueduct, and the fourth ventricle. Exposures in the posteroanterior, reverse Towne and lateral projections are then made. Sometimes, after this manipulation the fourth ventricle is incompletely filled and its floor may be obscured by a fluid level. If this occurs an alternative maneuver may be employed. After emptying the temporal horns by bringing the patient into the sitting position, he is taken through a very slow backward somersault until he reaches the inverted position. During this procedure the air bubbles slowly through the foramen of Monro into the third ventricle, aqueduct, fourth ventricle and out through the exit foramina of the fourth ventricle into the spinal subarachnoid space in the sacral region. If this maneuver is performed very slowly and provided there is no obstruction to the cerebrospinal fluid pathway, most of the air can be manipulated from the ventricles into the spinal subarachnoid space. If the patient is then brought into the prone or brow-down position and then very slowly into the sitting position with the head slightly flexed, the air will pass from the spinal subarachnoid space into the cisterna magna and gradually fill the ventricular system in the same way as during pneumoencephalography. This manuever will usually give complete filling of the fourth ventricle and aqueduct without a fluid level.

If, during ventriculography, it is desired to manipulate the air into the basal cisterns, the air is manipulated into the spinal subarachnoid space by the method described above and then the patient is brought into the brow-up or supine position. From this position he is brought very

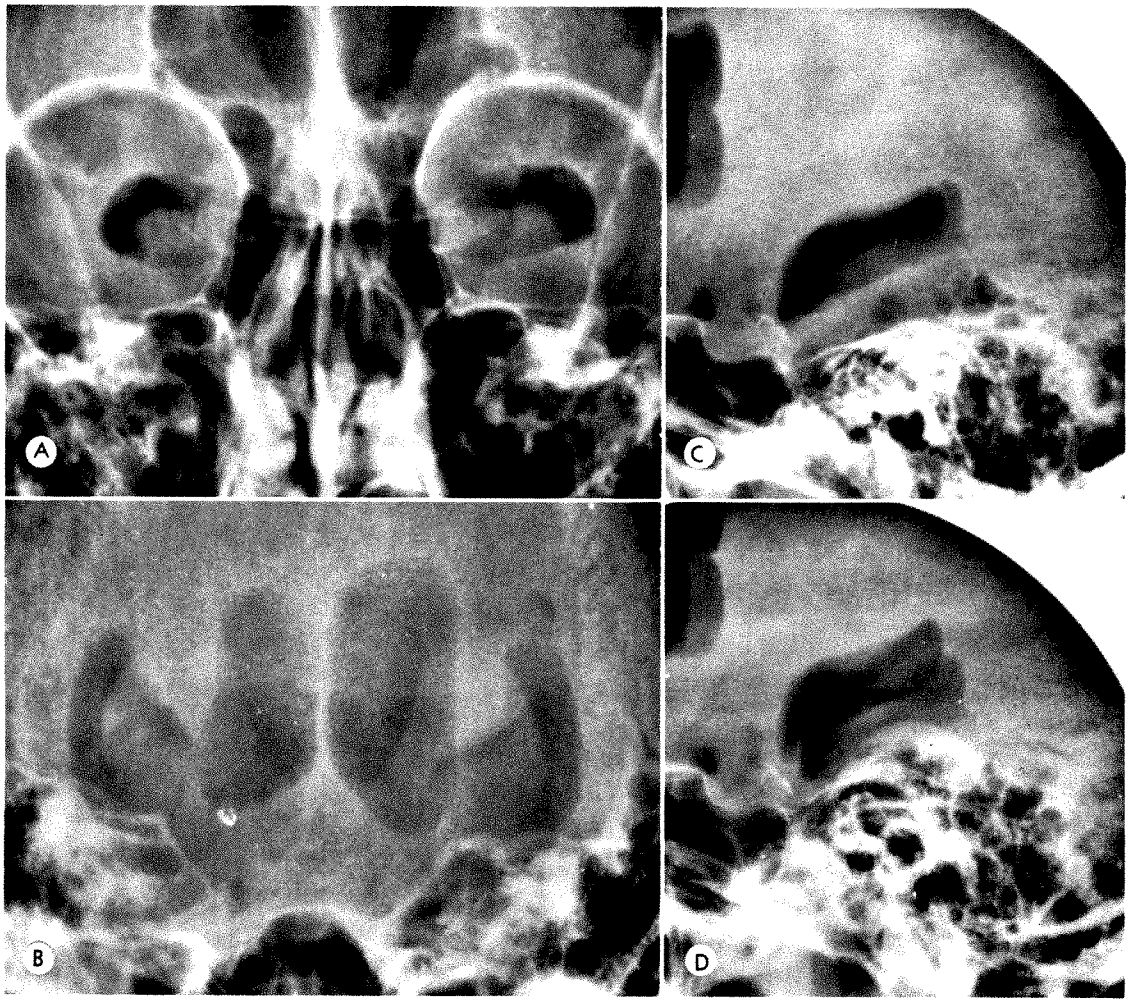


FIG. 4. Routine roentgenograms of temporal horns when both are filled simultaneously: (*A*) Orbital, (*B*) Towne, and (*C* and *D*) stereolateral studies.

quickly into the sitting position with his head extended. After about 10 seconds the head is flexed and roentgenograms are obtained. The burr holes for ventriculography may be placed in the operating room, but the ventricular puncture may be carried out in the radiology department with the patient in position in the somersaulting chair.

ADVANTAGES OF THE PRESENT TECHNIQUE

When lumbar encephalography is being performed, the needle may be left in place with the patient in any position. It is, therefore, possible to obtain complete filling of the entire ventricular system without air in the subarachnoid systems. At the conclusion of the ventricular study with the needle still in position, the subarachnoid spaces may be filled and studied. This is particularly valuable when a lesion in the suprasellar region is suspected. In such cases about 20 cc. of air may be injected into the ventricular system in the usual way and then the patient is slowly brought down into the supine or hanging-head position so that the anterior end of the third ventricle may be visualized. In this manner roentgenograms of the third ventricle may be obtained without air in the basal cisterns to obscure minor changes in this region. The patient then can be brought back



FIG. 5. Almost complete filling of the third ventricle during ventriculography in a case of aqueduct stenosis (arrow).

into the sitting position and the basal cisterns are filled by injecting more air with the head extended.

It has been found an advantage to have both temporal horns filled simultaneously so that the two sides may be compared, and this more than outweighs the disadvantage of having the two temporal horns partly superimposed on lateral roentgenograms. The chair has increased the speed with which air studies may be performed, particularly if polaroid films are used to check the degree of filling in the early stages. The somersaulting manuever is well tolerated by almost every patient and the method seems particularly valuable in cases where the patient has any type of respiratory difficulty. Such a patient may become quite cyanosed when lying in the prone position on a conventional support to obtain brow-down roentgenograms, but, when the chair is used, the weight of the body is taken on the shoulders and the chest is able to move freely. On rare occasions when lateral recumbency roentgenograms are required, the shoulder straps are loosened with the patient in the supine position and then the head can be brought into the necessary position without difficulty. The increased efficiency of air manipulation into different parts of the ventricular system by somersaulting methods enables satisfactory studies to be obtained with a smaller quantity of air than by conventional methods (Fig. 5 and 6, *A* and *B*). These methods have eliminated the need for the technique of central ventriculography recommended by Azambuja *et al.*[2] who placed a catheter directly in the enlarged third ventricle.

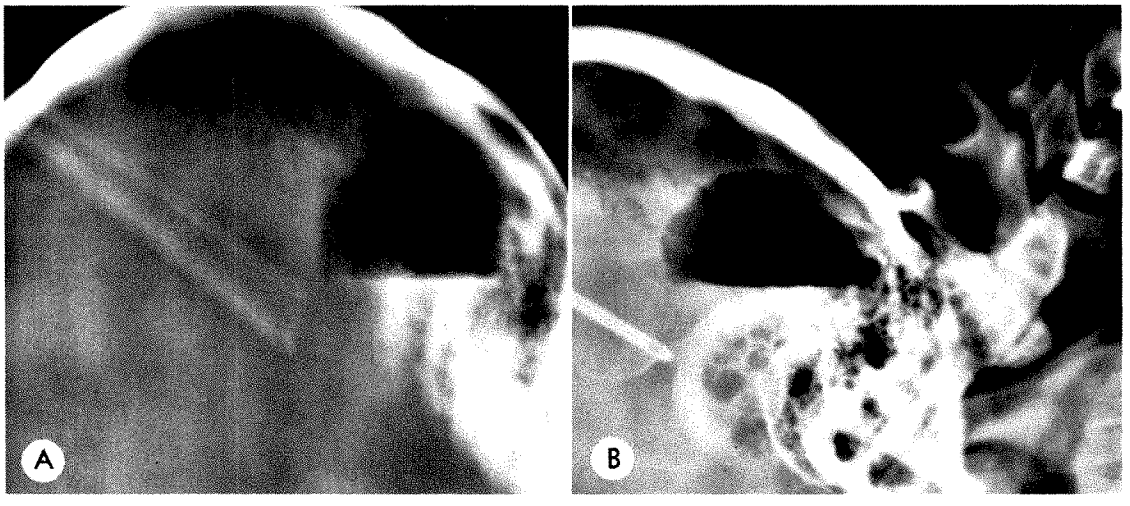


FIG. 6. Dandy-Walker syndrome demonstrated on ventriculograms. Only 20 cc. of air was introduced and all of the air was manipulated into the greatly enlarged fourth ventricle. (*A*) Brow-down lateral autotomogram and (*B*) forward hanging-head lateral study.

SUMMARY

A somersaulting chair for use in cerebral air studies is described. The manipulations required to demonstrate different parts of the ventricular system and the subarachnoid spaces are discussed. The advantages of this method of performing cerebral pneumography are presented.

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A MODIFIED ROTATING CHAIR FOR PNEUMOENCEPHALOGRAPHY AND VENTRICULOGRAPHY

By KENDRICK C. DAVIDSON, M.D.
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RECENTLY Amplatz[1] described a somersaulting chair for holding patients during pneumoencephalography which, because of the feature of 360 degrees forward or backward rotation, is particularly useful for re-positioning air in the ventricular system from the spinal subarachnoid space and for distributing air throughout the ventricular system itself. Use of the chair enables one to hold anesthetized or conscious patients easily during the entire procedure. Recently, a motor driven chair* was purchased with similar features which is patterned after the original chair developed by Dr. D. Gordon Potts and associates of Neurological Institute, New York, New York. This chair, like that of Amplatz, holds the patient securely and is capable of 360 degrees forward and backward rotation (Fig. 1 and 2), allowing the filling of various portions of the ventricles and subarachnoid cisterns.

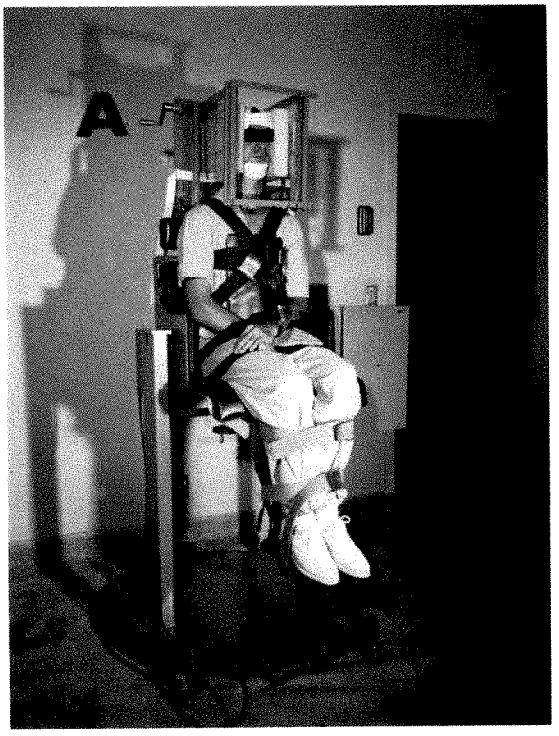


FIG. 1. Anterior oblique view of chair with an adult model in place. Crank is labeled "A."

The chair has been modified at our institution for use with infants, children, and adults. A pediatric chair is provided upon purchase of the main unit which can also be altered to advantage. Figure 3 shows the reinforced chair "A" and a specially built bracket "B" with slots of varying height in which the chair is secured beneath the head holder. Figure 4 shows the chair in place during an actual pneumo-

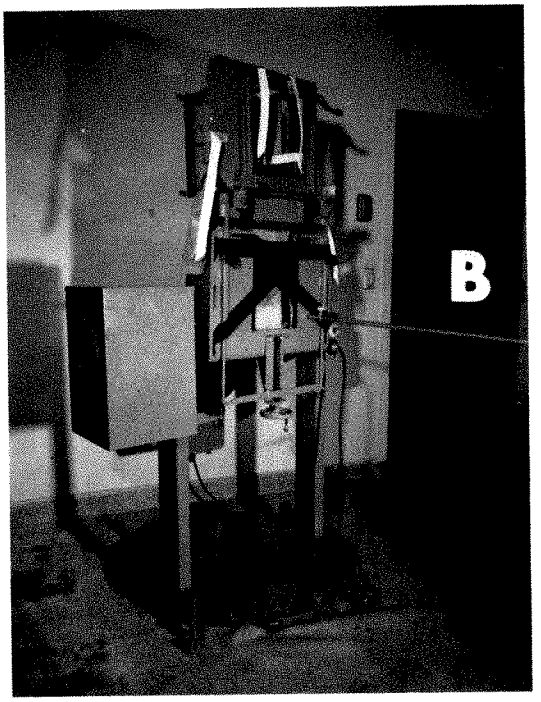


FIG. 2. Posterior oblique view of the chair. Detachable tomographic handle is labeled "B."

* The Mutural Corporation, Yonkers, New York. Available from Picker X-Ray Corporation, White Plains, New York.

encephalographic study of a 2 year old child under general anesthesia.

The transparent plastic head holding device has been changed from its original construction to allow fixation of the head between two height adjustable nontraumatic plastic sponges. For adult patients, final control of the head is achieved by an adjustable canvas webbing chin and forehead support. The entire back support and attached head holder unit are adjusted to the patient's height by a worm gear elevator operated from the back of the chair. The patient's skull is anchored securely in the head holder by adjusting the canvas harness, and by turning a single detachable crank labeled "A" in Figure 1, which operates the two lateral panels of the head holder through a gear and turn screw arrangement. Individual films are inserted for exposure in slots fixed to the various panels of the head holder. For this purpose, regular cassettes are used in combination with a thin, detachable, parallel line 8 to 1 ratio grid of 110 lines per inch construction. There is just room in each film slot of the head holder unit to accommodate a film and a grid, so that they stay in place when the patient is inverted in the chair. This arrangement overcomes the cost of grid front cassettes. Figure 5 is a routine upright anteroposterior pneumoencephalogram made of a 7 year old child showing dilated ventricles.

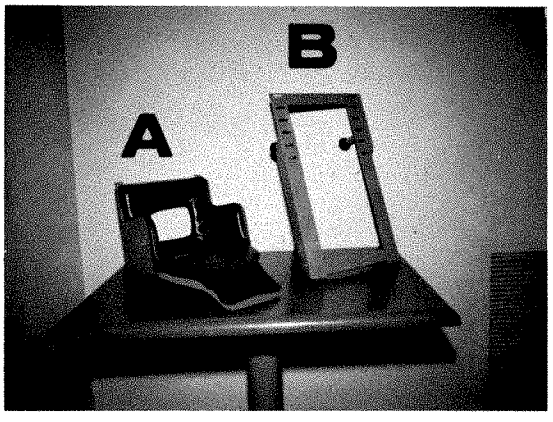


Fig. 3. Reinforced pediatric chair "A" and adapting bracket "B."

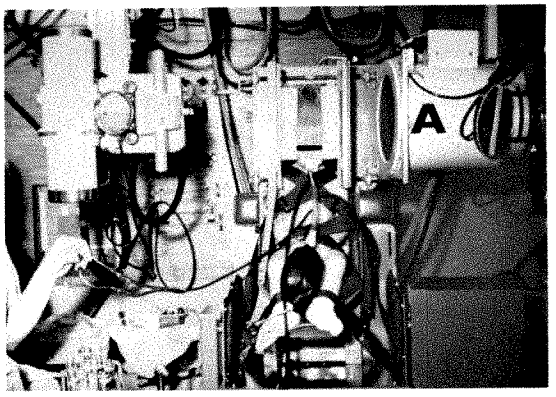


Fig. 4. The chair in use during pneumoencephalography of 2 year old child under general anesthesia. "A" is the image-amplifier used for visual monitoring of ventricular filling.

Four large door stops of heavy construction were added to further help anchor the chair base plate firmly during manipulation of the patient. With the base plate anchored, the chair swivels 360 degrees about a central axis on castor bearings, permitting

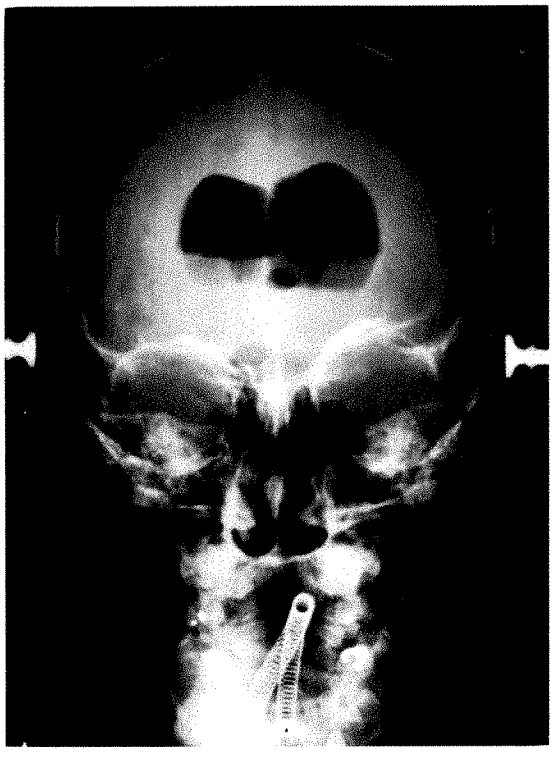


Fig. 5. A routine upright anteroposterior roentgenogram showing dilated air filled lateral and third ventricles in a 7 year old child under general anesthesia.



FIG. 6. An autotomogram of the midline, slightly dilated, air filled ventricles obtained by manual short arc rotation of the chair.

the production of autotomograms of midline structures during pneumoencephalography and ventriculography. For autotomography, the roentgen-ray tube is aligned to an upright portable or wall mounted cassette holder containing a parallel line grid front film. To produce tomographic movement, the chair is set in motion by grasping the long detachable handle, labeled "B" in Figure 2. It is moved horizontally through a continuous arc of 5 cm.; 2.5 cm. on each side of midline position. This maneuver is carried out during the first phase of ventricular filling. An exposure is made with the patient's head in neutral upright position, during turning of the chair, resulting in a sagittal autotomogram of the third ventricle, aqueduct of Sylvius and fourth ventricle sharply portrayed without overlying air filled structures, as shown in Figure 6.

Ventricular filling can easily be monitored visually during pneumoencephalography without the need for taking verification roentgenograms. This is done with an image amplifier-television system labeled "A" in Figure 4 which is energized by 1–2 second long exposures from the ceiling mounted roentgen-ray tube. For this purpose the roentgen-ray tube is operated at 70 kv. and 10 ma. This viewing procedure also eliminates retake roentgenograms, which are necessary if ventricular nonfilling occurs.

SUMMARY AND CONCLUSIONS

A modified, motor driven, stable 360 degree somersaulting pneumoencephalographic chair is described, which is adaptable to any age group patient, conscious or under anesthesia. Use of the chair greatly facilitates pneumoencephalography. The chair can also be used to advantage during ventriculography.

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The author wishes to express thanks to Mr. Harold E. Wirth, Master Machinist, for his assistance with the modifications of the pneumoencephalographic chair.

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Vol. 91, No. 5

A VISUAL EXAMINATION OF LAMINAGRAPHIC SYSTEMS*

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BODY-SECTION roentgenography is a special technique to visualize a predetermined body zone or plane while blurring or effacing parts of the object outside the plane of interest. Any comparative study of different laminagraphic methods should, therefore, be primarily concerned with the physical characteristics and limitations of the blurring pattern. There are many factors other than the moving tube-film system which affect the quality of any laminagram. Besio[1] lists approximately 900 articles concerned with the technical qualities of laminagraphy but no articles printed in English are specifically devoted to an appraisal of the different blur patterns. In view of the ever increasing number of body-section devices and methods, it seems appropriate that this primary quality be separately examined and its effect upon the body-section roentgenogram be determined.

The laminagram made with any device is a roentgen image of the focal plane plus three types of parasite shadows: *inherent blur;* the *incomplete blur* of parts of the object outside the focal point and those other extraneous shadows which are created by a *summation effect* within the blurring system. The present study is concerned with an examination of the basic blurring pattern of 4 laminagraphic systems, each of which is compared from 3 aspects: the significance of the blur, the origin and extent of the summation effect, and the influence of these physical characteristics upon the final laminagram.

BASIC BLURRING PATTERN

To examine the basic blurring pattern of any laminagraphic system and to separate the parasite shadows, one has but to remove the image of the focal plane and to create an image of any plane of the object outside of the focal plane, *i.e.*, to separate the blur from the focal plane image. To accomplish this end, a linear phantom was constructed of copper wire having a diameter of 0.010 inch or a thickness less than the minimum thickness of the thinnest section available in any laminagraphic system. Two identical phantoms were constructed (Fig. 1, *A* and *B*), one of which was permanently located in the plane of focus and the other was mounted on a micrometer scale having a 2 cm. excursion so that it could be positioned to represent any plane of the object outside of the plane of focus for a distance of 1 cm. on either side of that plane. The plane of focus and the other element planes of the object are then separate. Multiple roentgenograms of this phantom were made with 4 moving tube-film systems; namely, linear, elliptical, circular and hypocycloid. The resulting roentgenograms represent the basic blurring pattern of each laminagraphic system.

MOVING TUBE-FILM SYSTEMS

A general axiom of any laminagraphic system is that all parts of the object to be blurred which are perpendicular to the plane of section are maximally blurred or effaced while those parts of the object which are parallel to the plane of section are not blurred but are merely elongated. The ideal laminagraphic system then would be one in which the obscuring ray orginates from an infinite number of directions so that all parts of the object to be blurred would at some time be perpendicular to the obscuring ray and uniform blurring will result. Examination of the linear phantom with 0.5 cm.

* Department of Radiology, Guthrie Clinic Ltd., Robert Packer Hospital, Sayre, Pennsylvania.
Presented at the Sixty-fourth Annual Meeting of the American Roentgen Ray Society, Montreal, Quebec, Canada, October 8–11 1963.

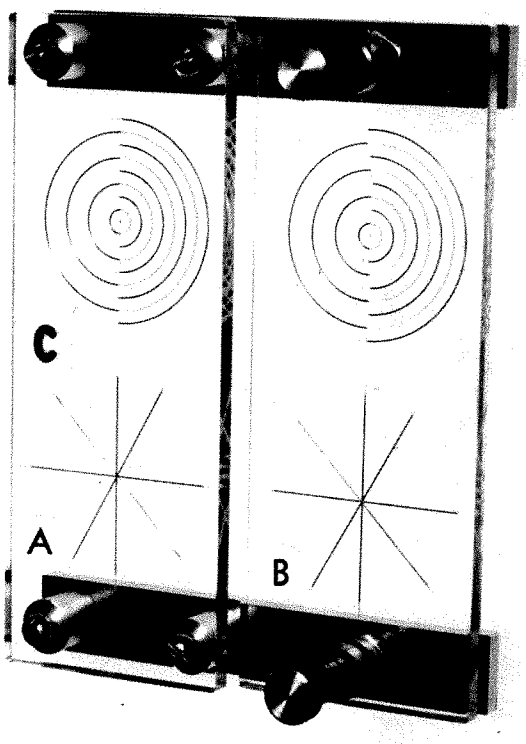


FIG. 1. Simple wire phantom with one pattern (*A*) located in the plane of focus and the other (*B*) on a micrometer scale so that it can be located at any level within 1 cm. above or below the focal plane. Locating phantom *B* outside of the plane of focus then separates the blur pattern from the image of the focal plane *A*. "C" in the subsequent figures represents the focal plane image.

of displacement employing a *linear movement* shows this to be true (Fig. 2, *A*, *B* and *C*). There is maximum blurring of those wires which are perpendicular to the obscuring ray, no blurring but merely elongation of the parallel wires with progressive blur of angled wires, and regularly progressive blur of the arcs. Further there is a difference in the blur pattern of the arcs of different radii. The blurring pattern of a linear laminagraphic system is a very irregular and incomplete blur. If we change the relationship of the phantom to the long axis of the obscuring movement, a different blurring pattern results which again is related to the obliquity of the object to the obscuring ray. Therefore, the blurring pattern created by a linear tube movement is, in effect, dependent upon the orientation of the object in the linear blurring system. The blur pattern will always reflect the predominant direction of the obscuring movement.

Progressing to an *elliptical movement* (Fig. 3, *A* and *B*), this latter feature is again reflected in the blurring pattern. With an ellipitical tube movement, an increased blur results because of the additional direction change of the tube-film blurring system but the basic linear elements still prevail.

A *circular movement* provides a very uniform blur (Fig. 4). However, at the margin

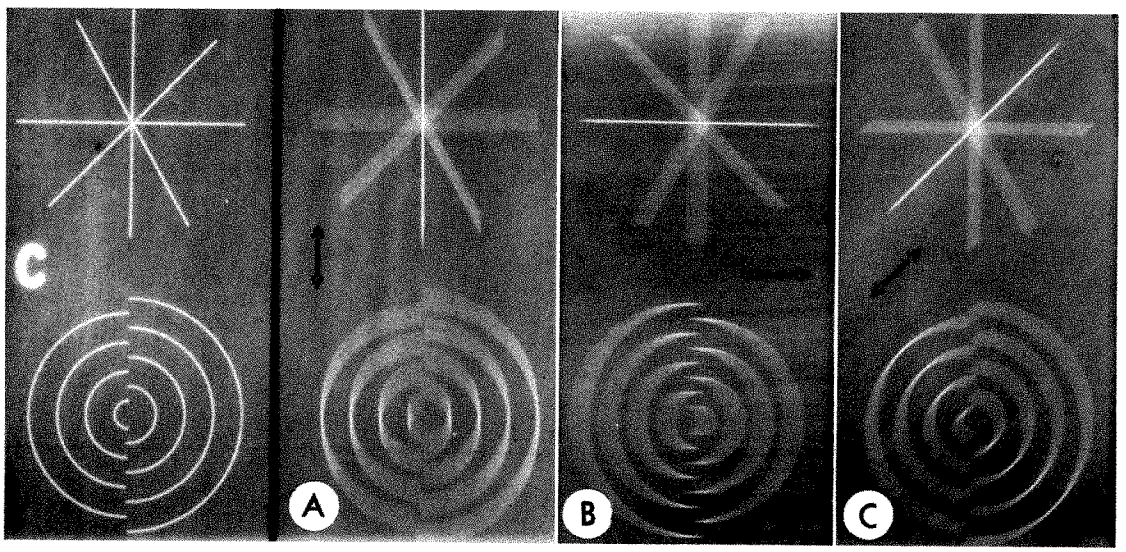


FIG. 2. The blur pattern of *linear* obscuring movements: longitudinal (*A*), transverse (*B*), and oblique (*C*). Focal plane image "C" on the left.

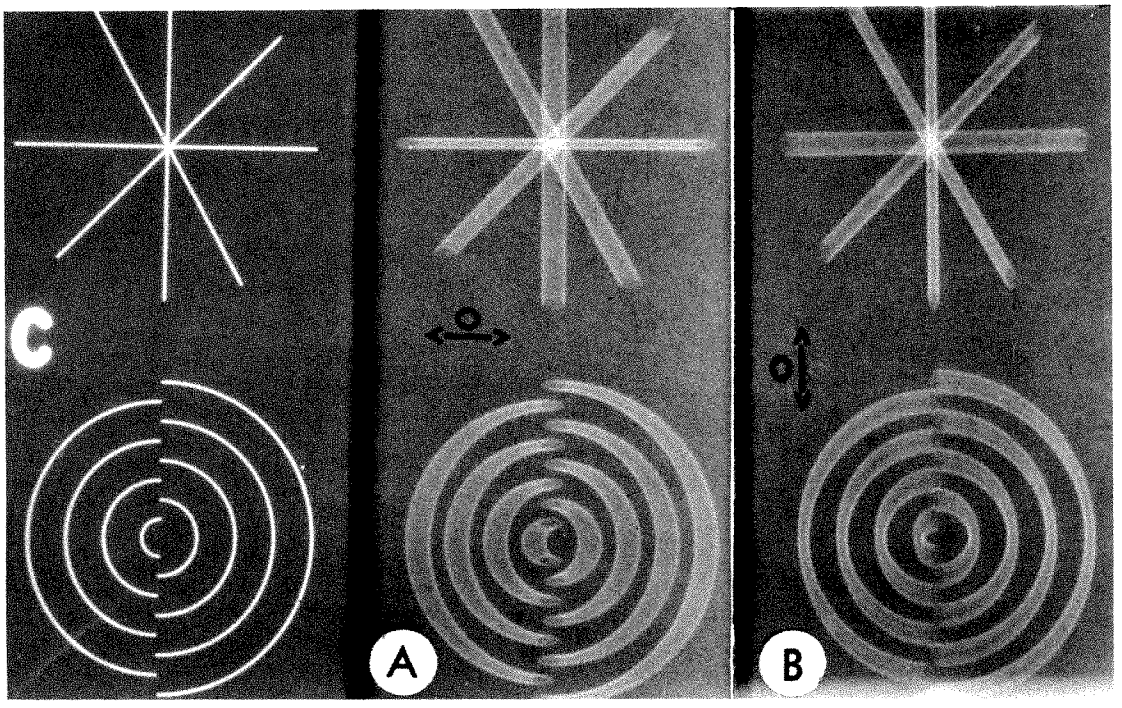


Fig. 3. The blur pattern of *elliptical* obscuring movements: transverse (*A*) and longitudinal (*B*). Note the irregular blur which results from the longitudinal element of this obscuring movement. Focal plane image "C" on the left.

of the blur a sharp cut-off line results and if the radius of the object to be blurred happens to coincide with the radius of the obscuring movement, a false shadow may be created by virtue of incomplete blur. As the separation increases, the blur pattern from a circular movement widens until the sharp cut-off margins fuse and create a pattern which is the mirror image of the object being blurred (Fig. 5, *A* and *B*). This phenomenon may represent what has been referred to by many authors as a parasite shadow created by a summation effect.

A uniform blur with a gradual fade at the margins of the blur pattern is seen with the *hypocycloid movement* (Fig. 6) which most closely approximates the ideal obscuring movement; namely, one which permits the obscuring ray to originate from an infinite number of directions.

The shortcomings of a linear system cannot help but produce factitious shadows in the plane of focus which distort the actual image and lead to false interpretations. If the mobile phantom is reversed and superimposed over the control at a level 0.5 cm. above the focal plane, then the simplest effect of any element laminagram is seen; namely, the influence of one element plane on the focal plane. In Figure 7, the focal

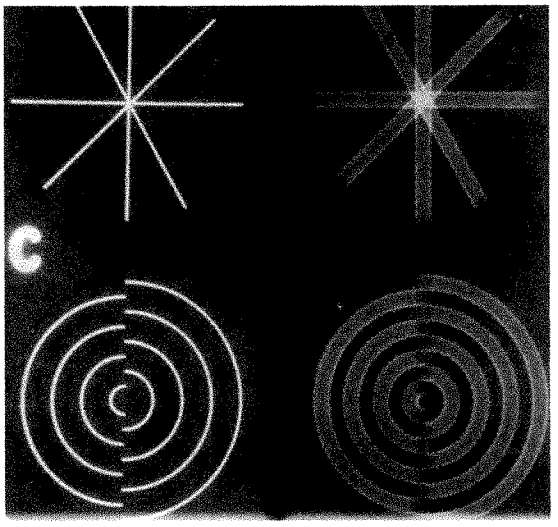


Fig. 4. *Circular* obscuring movement (36° circle). Note the uniform blur and sharp margin "cut-off" pattern. Focal plane image "C" on the left.

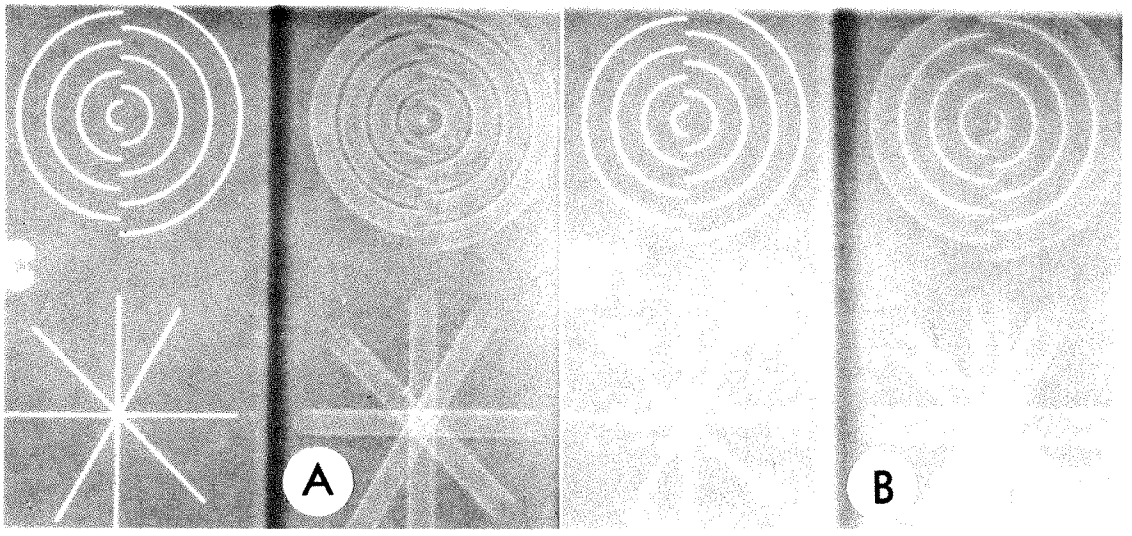


FIG. 5. (*A*) 36° circle, 0.5 cm. separation. (*B*) 36° circle, 1.0 cm. separation. As the separation is increased with a *circular* obscuring movement the sharp cut-off patterns fuse (*B*) to form the mirror image of the object to be blurred. "C" focal plane image.

plane image "C" is a laminagram made with the hypocycloid movement. An accurate image of the control is seen with the superimposition of minimal residual blur. The laminagram on the right exposes the pitfalls of linear laminagraphy. The incomplete blur of the element plane outside the focal plane creates a new and false geometric image.

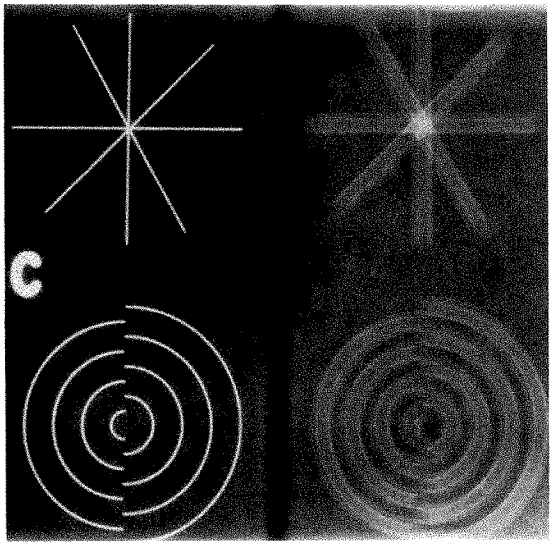


FIG. 6. *Hypocycloid* obscuring movement. Notice the uniform blur with an irregular blur extending out to the margins. Focal plane image "C" on the left.

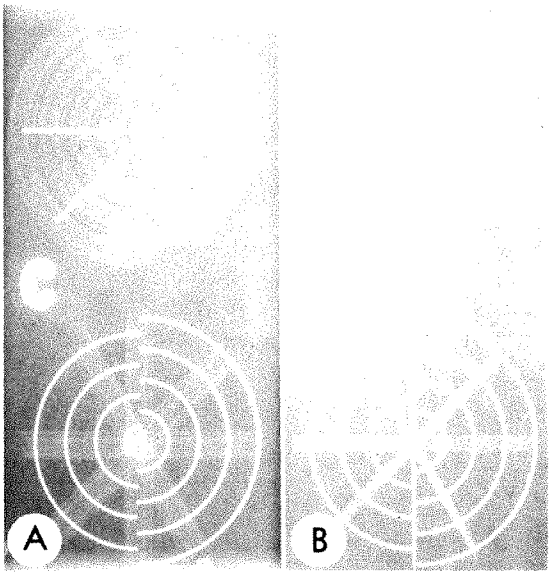


FIG. 7. Superimposition of the two wire phantoms, one of which is reversed and moved 0.5 cm. out of the plane of focus. The resulting image then equals the image of the focal plane plus the residual blur. With the *hypocycloid* movement (*A*), all elements of the phantom outside the plane of focus experience uniform blur and do not distort the image of the focal plane. With the *linear longitudinal* obscuring movement (*B*), however, a new geometric pattern is created in the focal plane as the result of the incomplete blurring of the object plane.

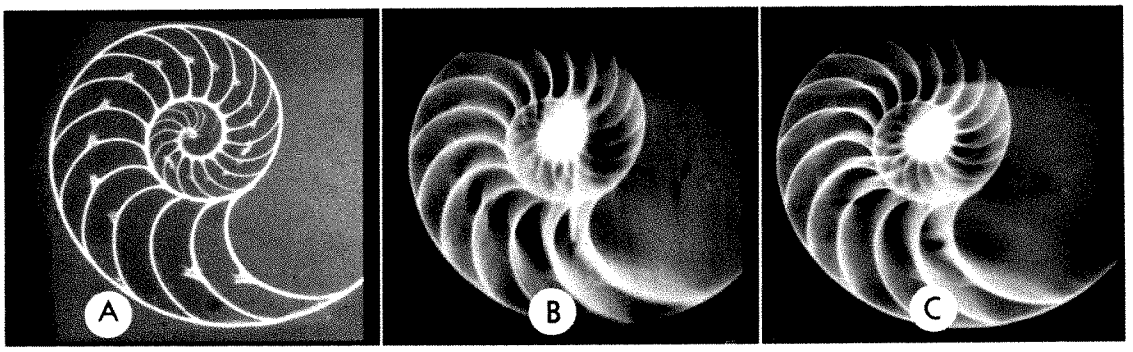


FIG. 8. The focal plane of the sea shell (*A*) was physically sawed out of the center of the sea shell and the above image of the *anatomic section* was obtained employing a 0° circle. The *linear longitudinal* (*B*) and the *linear transverse* (*C*) movements demonstrate the incomplete blurring of some septa and the creation of factitious shadows in the central spiral.

Summarizing, the direction of the tube-film movement in any laminagraphic system is reflected in its blur pattern. A linear obscuring movement results in an unequal and irregular blur, an elliptical movement produces increased but again unequal blur. A uniform obscuring results from circular movements but this tube-film movement is prone to create summation shadows which result from the fusion of sharp cut-off shadows at the margins of this blur pattern. The hypocycloid movement with an angle of section of 48° produces the greatest and most irregular blur with the lowest potentiality to present residual blur or to create false shadows in the plane of interest.

A body-section roentgenogram can thus be regarded as containing an infinite number of laminagraphic planes each providing element laminagrams all of which are integrated in the final laminagram. A body-section layer may be defined, therefore, as that stratum containing all those laminagraphic planes from which element laminagrams are produced that contribute to the sharpness of the final image.[2]

VISUAL DEMONSTRATION

To visually appreciate the effect of any laminagraphic system upon any element plane outside the plane of focus, the movable plane of this linear phantom was positioned 1 cm. outside the plane of focus and moved into the focal plane and out for a distance of 1 cm. on either side at 0.2 mm. increments. A roentgenogram made at each level was then copied on 16 mm. film and a projection of this film strip provided a visual appreciation of the foregoing principles.

SEA SHELL PHANTOM

For practical purposes, however, laminagraphy is always concerned with 3 dimensional objects and, hence, with the superimposition of infinite numbers of blur patterns, some of which may perform an obscuring function of their own. To examine this possibility a phantom of 3 dimensions consisting of simple geometric design, yet of regular irregularities was chosen; namely, a sea shell of the genus Nautilus. The equiangular spiral of this sea shell demonstrates multiple arcs and direction changes of thin bony structure simulating the complexity of bony trabeculae yet with a simplicity which permits comparative examinations.

A laminagram of the mid-portion of the sea shell was made with each of the 4 obscuring movements and the resulting laminagrams were compared to a plain roentgenogram of the focal plane which was physically sawed out of the center of the sea shell (Fig. 8*A*). The linear longitudinal laminagram (Fig. 8*B*) shows numerous extraneous shadows resulting from the incomplete blur, and in the most central portion of the spiral, which unfortunately is the most difficult part of the sea shell to

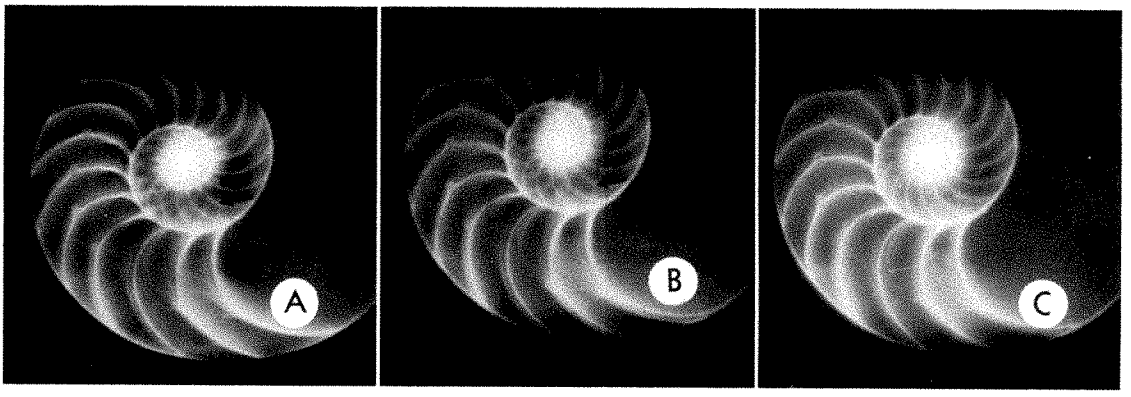


FIG. 9. The *elliptical* obscuring movement (*A*) gives a more uniform blurring; however, note the factitious shadows which are undoubtedly the summation shadows resulting from the fusion of the cut-off margins of the circular component of this obscuring movement. A *circular* (36° circle) obscuring movement (*B*) produces the same effect; some of the septa carry a wider blur margin than others, probably incomplete blurring, because of coincidence of the radii of the septa and the obscuring movement. The *hypocycloid* obscuring movement (*C*) presents uniform blurring with no factitious shadows.

roentgenograph, spiral septa appear in the laminagram, although, as evidenced from the anatomic section in Fig. 8*A*, they were physically absent. Therefore, this obscuring movement has created factitious shadows in the focal plane which can lead to a misrepresentation of the actual structures of the focal layer. Similar but different shadows were created by changing the direction of the tube to transverse linear movement (Fig. 8*C*). Thus, it does not seem likely that by using two linear movements the extraneous products of the linear system would be reduced, rather they would tend to increase the total number of parasite shadows.

The elliptical movement would appear to give a better roentgen image of the focal plane (Fig. 9*A*), but again it can be noted that, in the middle of the spiral, septa are seen running in both directions. Comparing these to the anatomic section (Fig. 8*A*) it may be reasonably assumed that the circular components of the elliptical tube movement have resulted in a summation effect which has produced mirror images of these septa. The same phenomenon is prevalent only more pronounced in the circular movement (Fig. 9*B*). The hypocycloid movement gives the most reliable roentgen image of the actual plane of focus with a minimum of extraneous shadows (Fig. 9*C*).

HUMAN SKULL PHANTOM

Although the blur patterns examined above are interesting, they may not necessarily be of clinical importance. Perhaps the summation of the parasite shadows of all the element laminagrams has a canceling effect so that in the final laminagram the summation of the residual blur and the factitious shadows may not be as significant as considered thus far. Ziedses des Plantes[3] suggests that the image formations of anatomic areas are not comparable to simple test objects and that the canceling effect may be noteworthy. To examine this possibility a human skull was embedded in plastic and sectioned into multiple coronal layers each 2 mm. thick. The skull was then re-assembled with the addition of fiber spacers to accommodate for the loss of bone created by the saw cut. A plain plastic phantom was constructed of opal plastic dummies of each layer (Fig. 10, *A* and *B*). Then by substituting the dummy plastic layers for anatomic layers, the influence of the blur pattern of any element section on the resulting laminagram could be studied. For example, only the anatomic section number 19, approximately through the posterior portion of the hard palate and the optic foramina, was chosen as the layer of focus and successive layers were added one

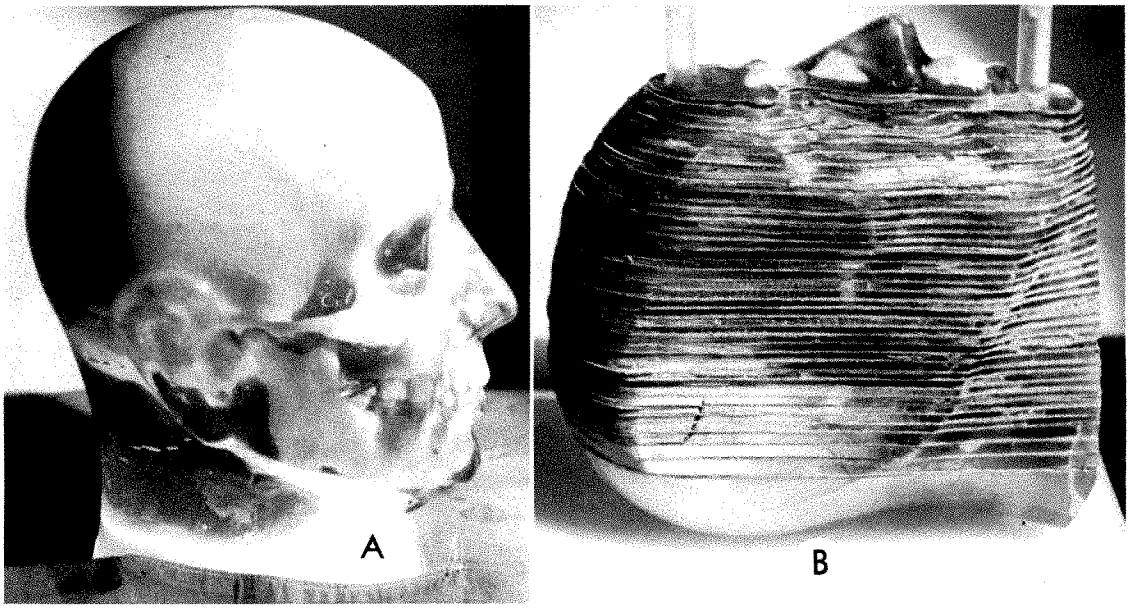


Fig. 10. A human skull (*A*) was sectioned into 2 mm. increments and re-assembled (*B*).

at a time until this portion of the skull was completely reconstructed. The influence of the summation of the element laminagrams was then noted. Three films of each series were selected for photographic comparison. In the linear longitudinal movement (Fig. 11, *A*, *B* and *C*), even the addition of the first layer shows the influence of the incisor teeth and nasal septum. When one-half of the element layers is added, this effect becomes more significant and when the entire group is added, the linear effect is very striking and obscures and distorts much of the detail in the mid-portion of the laminagram. When the tube movement is turned 90° to a transverse direction (Fig. 12, *A*, *B* and *C*), the linear effect is significantly less because most of the structures to be blurred are now perpendicular to the plane of section. Incomplete blurring of those portions of the object which are parallel to the plane of section can still be seen; for example, the sphenoid ridges. An elliptical tube movement (Fig. 13, *A*, *B* and *C*) shows a very decided improvement in the residual blur but again even in the first laminagram the shadows of the teeth are showing and in the final laminagram the laminations of the phantom can be very clearly picked up. These again represent the summation effect that was described earlier. The circular tube movement shows a better obscuring effect (Fig. 14, *A*, *B* and *C*). The teeth in Figure 14*A* are only vaguely visualized and Figure 14*C*, when all the layers have been added, is quite good. In the final series employing the hypocycloid movement (Fig. 15, *A*, *B* and *C*), the teeth are not recognized as such in any of the layers and the summation effect of the laminations of the phantom is somewhat less than it is with the circular movement.

SUMMARY

The composition of a body-section roentgenogram is a roentgen record of the shadows of the normal anatomic and pathologic structures of the selected layer plus the parasite shadows resulting from the laminagraphic system. The form and extent of the latter shadows fall into two classifications; *i.e.*, those which represent incomplete blur of neighboring structures and those which are created by the summation of factitious shadows in the laminagraphic system.

The effect of the factitious shadows upon the resulting roentgenogram is related to the

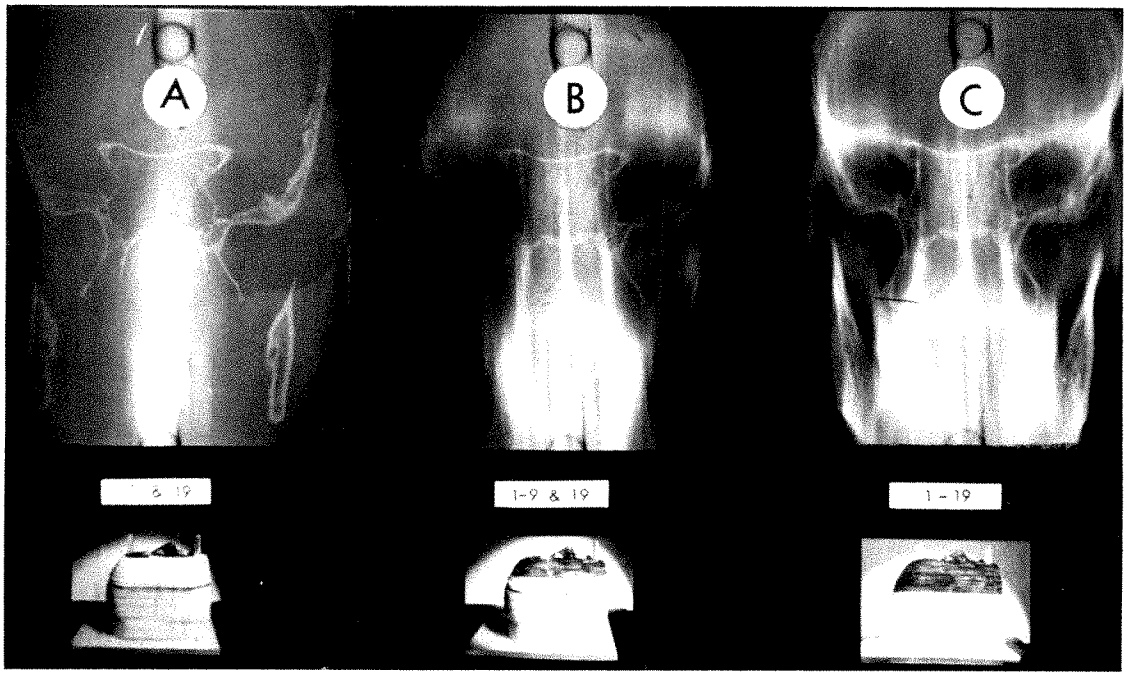


FIG. 11. Three laminagrams of phantom skull employing a *linear longitudinal* obscuring movement. *A* is composed of only 2 planes: the plane of focus 19 and one element plane "1". *B* includes 9 element planes 1–9 and the focal plane 19. *C* was made with all of the element planes 1–19. The incomplete blur of the first element layer persists and, as a matter of fact, is augmented throughout the succeeding sections.

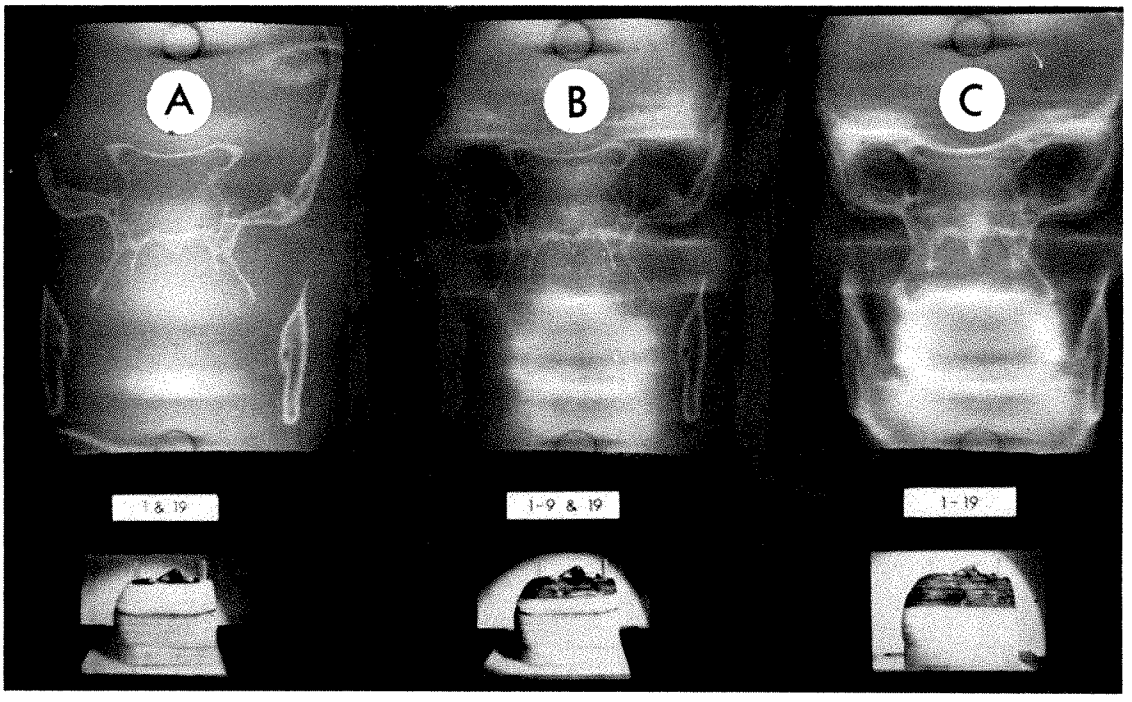


FIG. 12. (*A*, *B* and *C*) A *linear transverse* obscuring movement demonstrates fewer extraneous shadows because the major bony elements to be obscured lie perpendicular to the plane of section. There is still incomplete blur of parallel bony elements, for example, the sphenoid ridges.

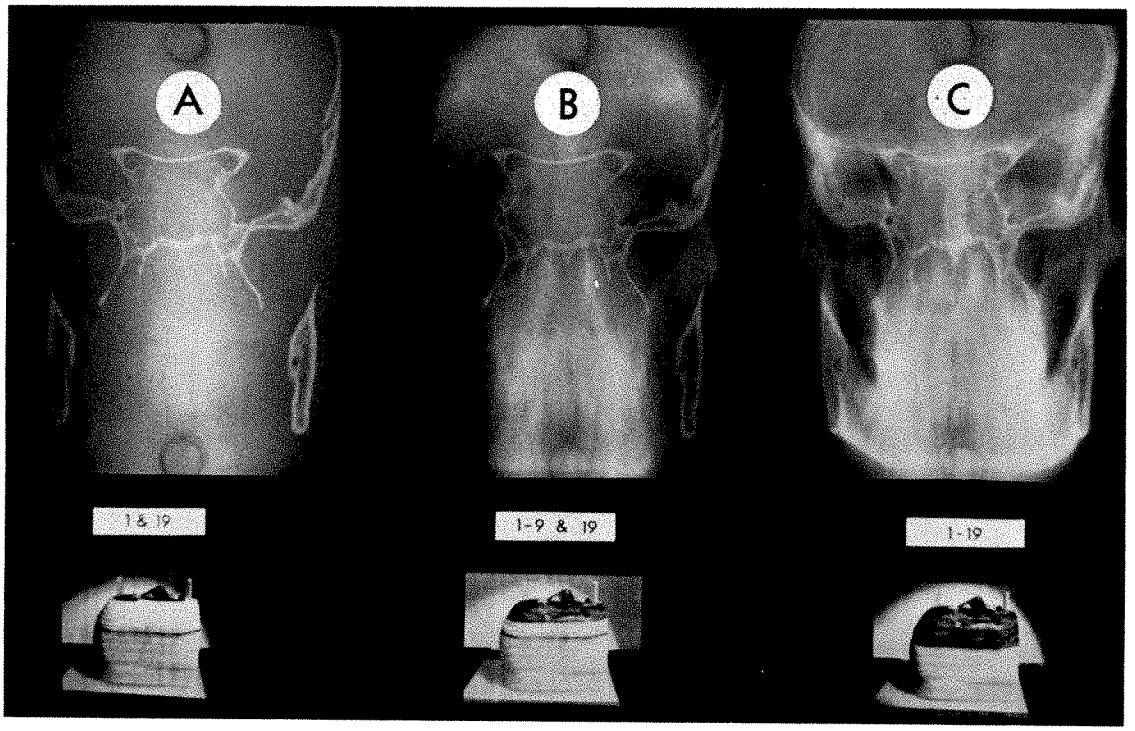


FIG. 13. (*A*, *B* and *C*) An *elliptical* obscuring movement shows more complete and more uniform blur. The striated effect seen in *C* is probably a summation effect arising from the superimposition of the shadows of the laminations of the phantom skull.

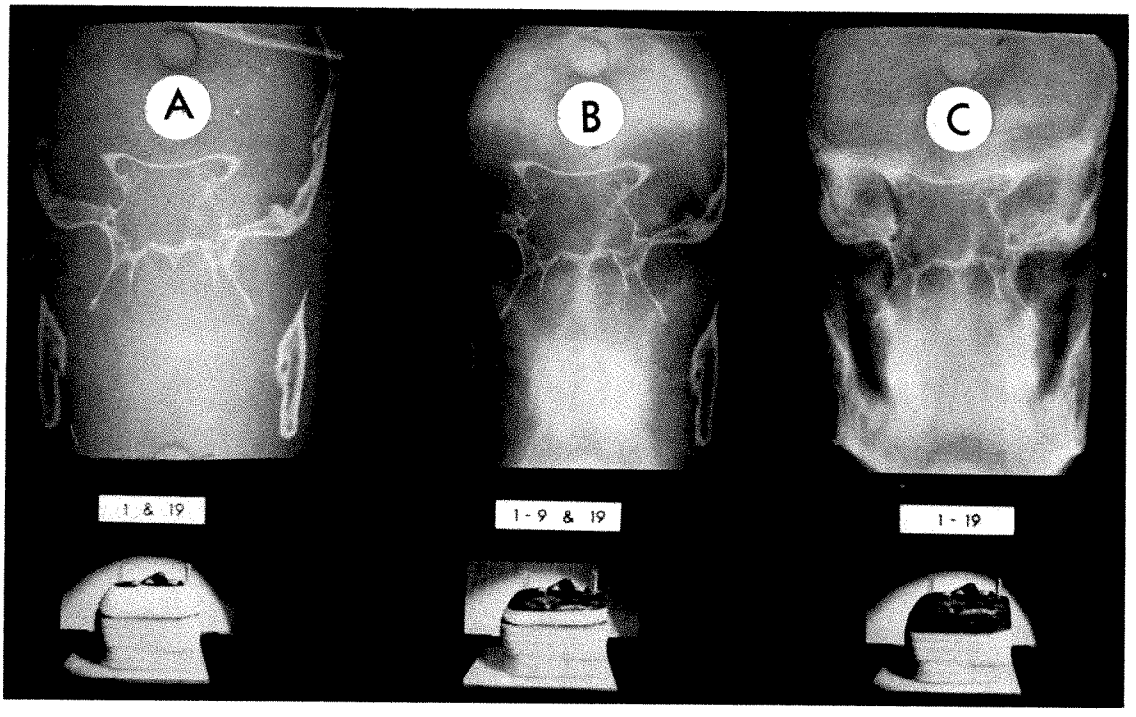


FIG. 14. (*A*, *B* and *C*) A *circular* (36° circle) obscuring movement effects more complete blur of element layers; however, the laminations in *C* resulting from a summation effect are more pronounced than with the elliptical movement.

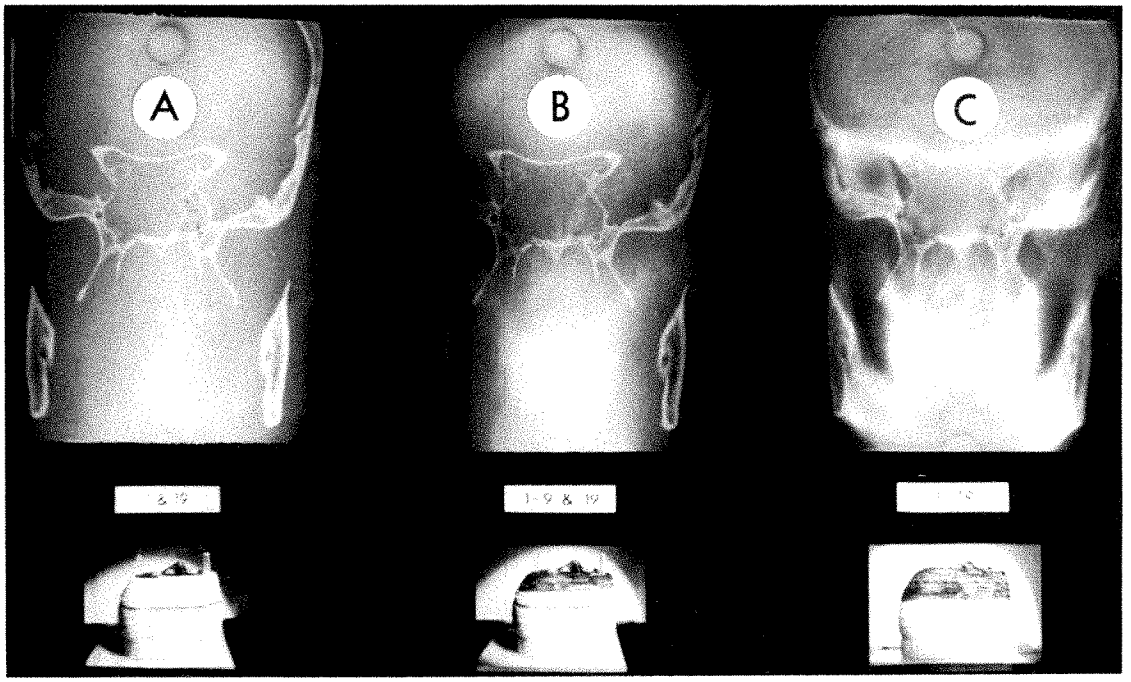


FIG. 15. (*A*, *B* and *C*) A *hypocycloid* obscuring movement with uniform blur. The summation effect is not as marked as it was with the circular movement.

function of the obscuring movement. The more closely the moving tube-film system corresponds to the theoretic ideal situation of having the obscuring rays arise from an infinite number of directions, the better is the resulting laminagram. The results of the 4 obscuring movements studied would indicate that the hypocycloid movement produces the most complete blur and the fewest summation shadows to give a resulting laminagram of high quality. The circular tube-film movement is next, to be followed by the elliptical movement, and from this study the usefulness of linear obscuring movements in any direction is certainly limited.

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DIAGNOSTIC APPLICATIONS OF CLOSED-CIRCUIT TELEVISION IN NEUROROENTGENOLOGY*

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THE application of closed-circuit television techniques can extend and enhance the diagnostic qualities of some roentgenographic examinations in a rather dramatic manner.[1] The present investigations, while representing only the initial efforts in this regard, seem to indicate that television should not be limited merely to its application as a medium of communication.

For years, as newer photographic techniques have been developed, they have been applied in an effort to improve the diagnostic features of roentgenograms. Now that the television image has been improved sufficiently to show smaller details, it seems only reasonable that radiologists should make use of some of its unique capabilities, such as the alteration of density and contrast, image reversal and magnification. All of these changes may be accomplished rapidly through electronic means rather than through the tedious and time-consuming maneuvers of existing dark-room photographic techniques.

During the installation of a closed-circuit television linkage between the x-ray department on one floor and the neurosurgical operating rooms on another floor of St. Mary's Hospital, Rochester, Minnesota, the idea occurred that such an apparatus might be adapted to the "subtraction read-out" technique, in which interest has recently been revived because of its value in some examinations using contrast materials.

Accordingly, the manufacturer of the closed-circuit television system was asked to provide two cameras with which to scan two roentgenograms and to apply these images simultaneously to a single monitor after electrically reversing the signal in one camera. A single synchronization circuit was to be used by both cameras, and controls were to provide for the image of one camera to be electrically enlarged or contracted as well as moved horizontally or vertically in order to supply as nearly perfect matching of the two images as possible.

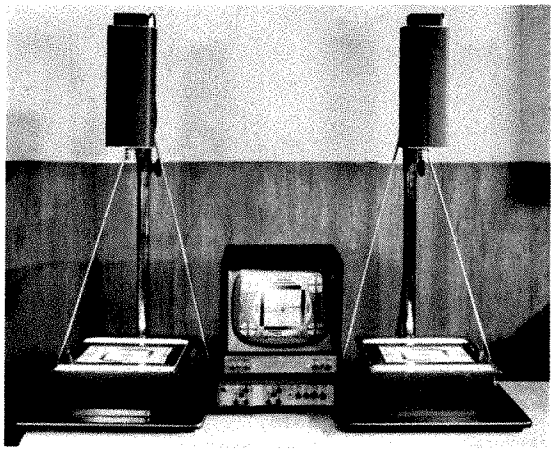


FIG. 1. Television apparatus for roentgenologic "subtraction read-out" technique.

Figure 1 shows the original apparatus and the vertical mounting of the two television cameras on stands which are ordinarily used for photographic enlargement apparatus. The cameras are optically focused on the two ordinary view boxes below, and the monitor with its controls is placed in the center between the view boxes. The monitor is of the regular 525-line-scan closed-circuit television type.

The chief advantage of this technique is the rapidity with which satisfactory subtraction studies may be accomplished. Occasionally, a vascular lesion can be identified in the television subtraction study even though it is not evident on the original roentgenogram (Fig. 2, *a*, *b* and *c*). This is not to say that such a shadow could not be observed at all; but because of overlying

* Presented at the Sixty-fourth Annual Meeting of the American Roentgen Ray Society, Montreal, Quebec, Canada, October 8–11, 1963.

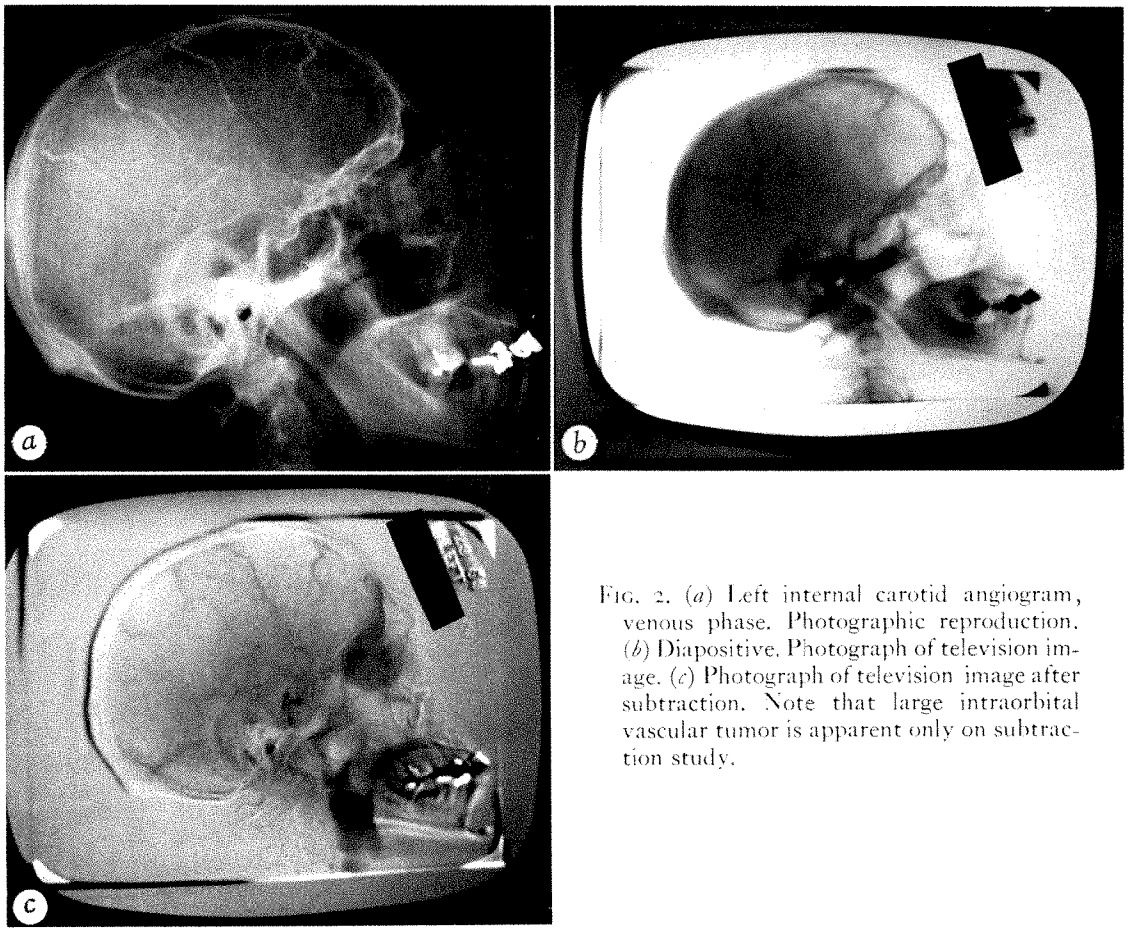


FIG. 2. (*a*) Left internal carotid angiogram, venous phase. Photographic reproduction. (*b*) Diapositive. Photograph of television image. (*c*) Photograph of television image after subtraction. Note that large intraorbital vascular tumor is apparent only on subtraction study.

obscuring shadows, it could not be identified for what it actually represented.

Experimental studies using a water phantom, an aluminum step wedge and dried bones, together with contrast-filled plastic and glass tubes indicated that to be seen in the subtracted image the contrast material in a tube of any practical size must be concentrated enough to be apparent on a good-quality roentgenogram without the subtraction technique. Thus, while one may frequently identify a lesion on re-examination of the original roentgenogram, this technique more easily identifies the process in the first place and makes the radiologist very much more confident of the diagnosis in nearly all cases.

The same apparatus is used to change the density and contrast, within limits, of roentgenograms of unsatisfactory quality. It is common practice to use a bright spot light when examining a very dark film. In some studies in which the roentgenographic image is too faint, it is possible to improve the quality of the picture by viewing it on the monitor after adjusting the brightness and contrast controls (Fig. 3, *a* and *b*).

In some instances there seems to be some subjective improvement in the study of examinations employing contrast medium by reversing the image so that a positive rather than a negative image is obtained. This is the customary method of presentation used by many fine European journals devoted to roentgenologic subjects and is readily accomplished by the use of the closed-circuit television camera in which the signal has been electrically reversed.

In order to magnify the image, two large lenses are applied to the television cameras and in this manner either a magnified roentgenogram or an enlarged subtraction study

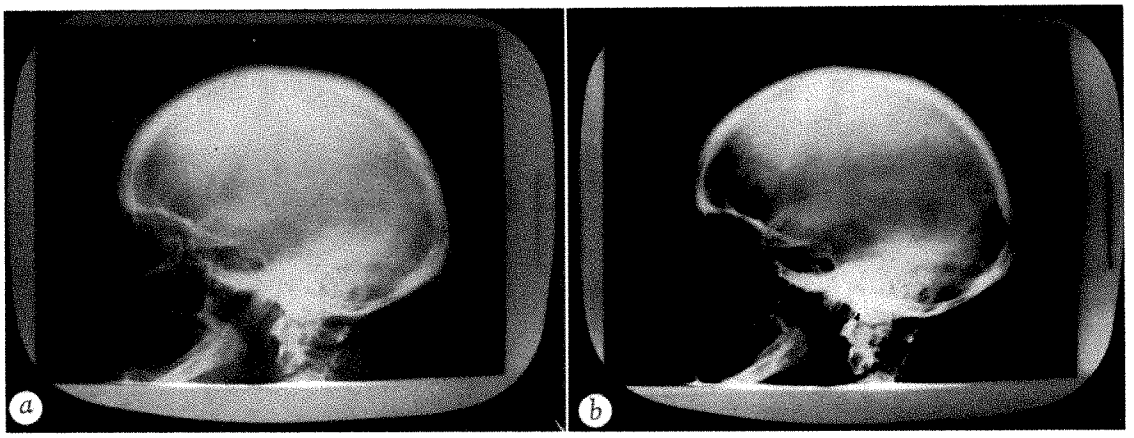


Fig. 3. Same roentgenogram of the head showing (*a*) increased brightness and (*b*) increased contrast by manipulation of the television controls.

may be obtained (Fig. 4).

A record of the television image can be obtained by photographing the monitor if desired (Fig. 5). However, if the roentgenograms are available, each study can be quickly re-examined with the machine at a moment's notice.

By way of projecting the use of this technique into the future, a study is now underway applying the system to cineroentgenographic examinations using opaque medium. Because it is imperative that there be virtually no motion of the organs examined between exposures of the two films, it is difficult to obtain good subtraction studies of lesions in the abdomen and chest. Cardiac, respiratory and gastrointestinal motion all tend to interfere.

In order to obviate this difficulty, cine examinations are performed in the usual manner at about 30 frames per second. There is, therefore, only a very slight interval of time between a frame showing no opaque medium in the vessels of an organ and the first ones showing the perfusion.

An apparatus is being constructed to project these two images simultaneously into the lenses of the two television cameras by means of a double, synchronized 16 mm. projector in such a manner that when using one film strip, one camera lags the other with respect to the film sequence by whatever interval seems appropriate to subtract all but the advancing bolus of contrast material. A project using video tape recording in the same manner is also now under investigation.

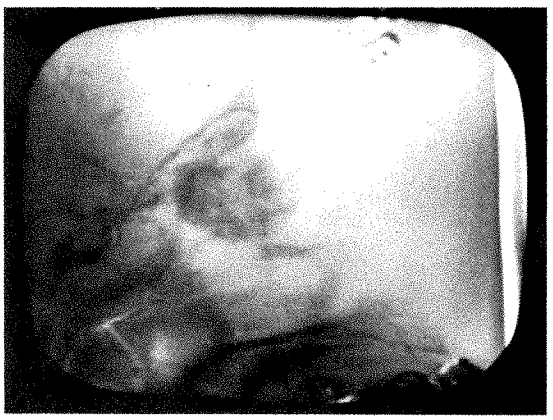


Fig. 4. Magnified image of the orbital tumor. Same case as in Figure 2, *a*, *b* and *c*.

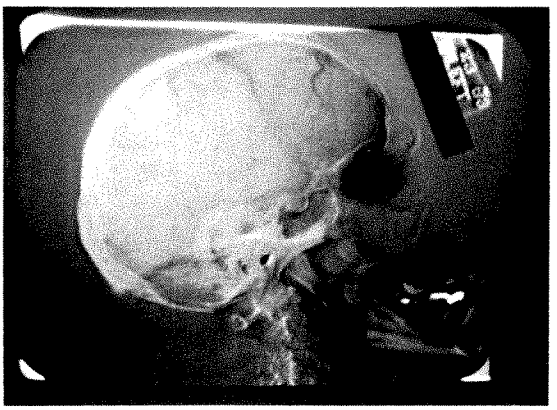


Fig. 5. Polaroid print of television image seen in Figure 2*b* made in 10 seconds.

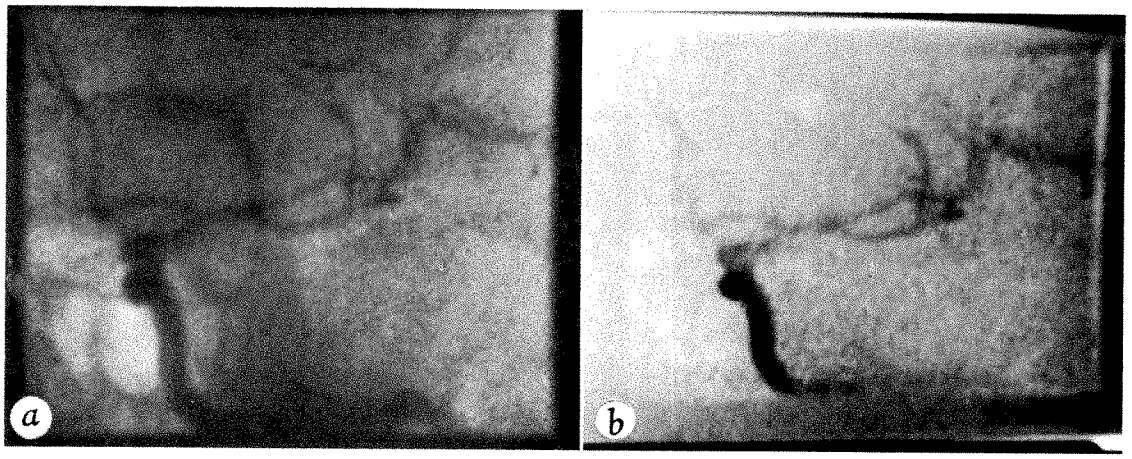


FIG. 6. (*a*) Sixteen millimeter cine frame of cerebral arteriogram. Oblique view through left orbit. (*b*). Subtracted image of the same 16 mm. cine frame as in *a*. Graininess of picture is due to the very great photographic enlargement before application to television apparatus.

In order to test this suggestion, two separate frames of a cine study of a cerebral angiogram (Fig. 6, *a* and *b*) and of an abdominal aortogram (Fig. 7, *a* and *b*) were greatly enlarged photographically and applied to the present apparatus in the usual manner. Although the resulting enlargements were necessarily very grainy in appearance, a subtracted image free of the overlying bony shadows was obtained. The photographic enlargement step will not be necessary where cine images are projected directly onto the television camera. It is anticipated that this technique may provide a useful and practical procedure for contrast examinations in organs in which such procedures have been disappointing up to the present time.

It is difficult to say at this time just how valuable this type of an apparatus will become. It certainly improves some roentgenographic examinations; and with further development it may provide yet another tool in the ever-increasing scope of diagnostic roentgenology.

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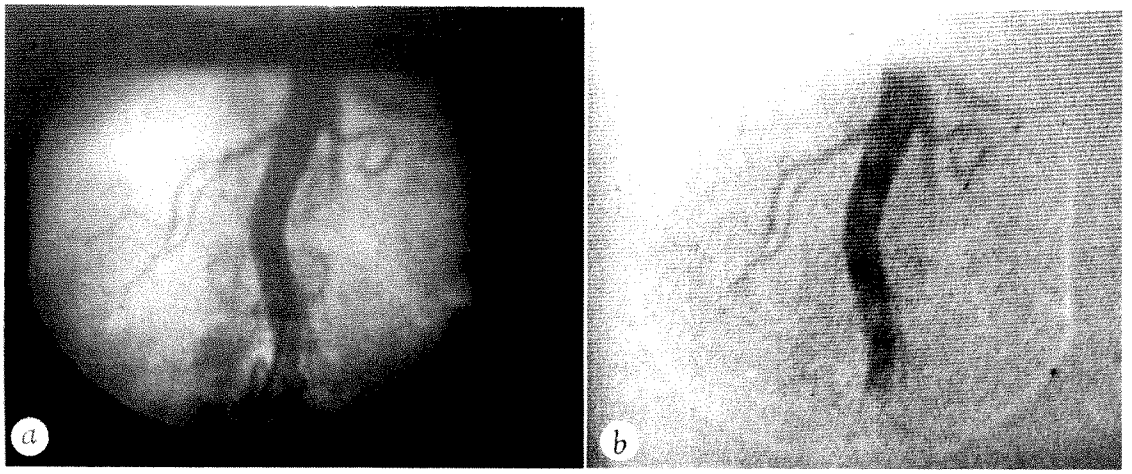


FIG. 7. (*a*) Sixteen millimeter cine frame of abdominal aortogram. (*b*) Subtracted image of same 16 mm. cine frame as in *a*. Here again graininess is due to very great photographic enlargement.

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EWING'S SARCOMA*

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DESPITE long established debate among histopathologists, most authorities now believe that Ewing's tumor is a distinct clinical pathologic entity and is not a variant of reticulum cell sarcoma. This point is important since prognosis rises from less than 5 per cent five year survival with Ewing's tumor to approximately 50 per cent with reticulum cell sarcoma. Comprehensive data are available regarding survival rates, but no significant statistical data were published in regard to local recurrence or visceral and bony metastases. The clinical aspects, roentgen and microscopic characteristics, and methods of therapy have been thoroughly described and will not be repeated herewith.

A case is reported to emphasize the following points:

1. Seven year survival is rarely encountered in histologically confirmed Ewing's sarcoma with roentgen and clinical evidence of local recurrence 1 year following initial radiation therapy plus pulmonary metastases 18 months following initial treatment.

2. Radiation therapy of local recurrence should be seriously considered, and, at least in this instance, has proven justifiable.

3. Pulmonary metastases do not necessarily indicate an early fatal outcome.

REPORT OF A CASE

This well developed and nourished 7 year old Irish male entered the hospital on September 1, 1956 complaining of pain in the left lower leg of 2½ months' duration. In June 1956 he was kicked on the lateral aspect of the left leg below the knee by his younger brother and subsequently developed a progressive limp. Roentgenograms at that time (Fig. 1) were interpreted as simple fibular fracture without displacement; no treatment was instituted. His limp became pronounced and the previously noted pain was progressive. He was therefore referred by his family physician for orthopedic consultation.

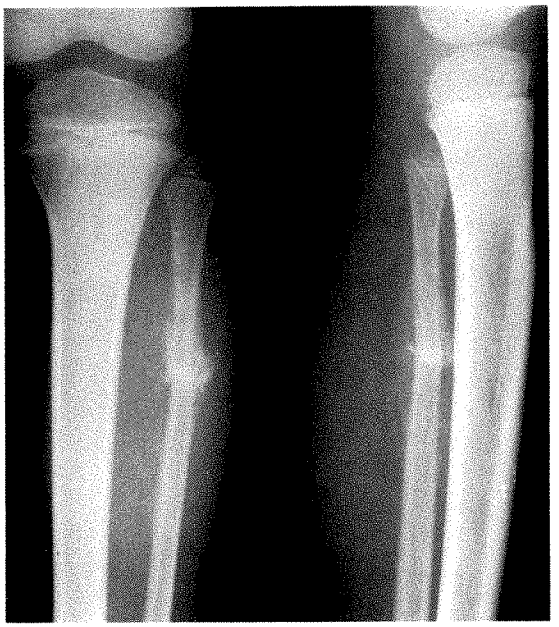


FIG. 1. Initial studies of the left fibula in June, 1956 demonstrating the fracture of the proximal shaft. Close inspection reveals considerable periosteal reaction and perpendicular radiating spicules above and below the fracture site.

Positive physical findings were limited to the left lower leg which was larger than the right (by 2.5 cm. in circumference at the level of the lesion). There was a sensation of warmth over the lateral aspect of the left upper leg with tenderness to palpation. The sedimentation rate was 48 and the temperature during his early hospital stay varied between 99° and 100° F. Chest and other bone survey revealed no roentgenographic abnormality. All of his laboratory studies other than the sedimentation rate were normal.

Incisional biopsy of the lesion on September 4, 1956 revealed a reddened, thickened periosteum surrounding the lesion. Considerable grayish pus exuded from the periosteal incision. The clinical appearance at surgery was that of

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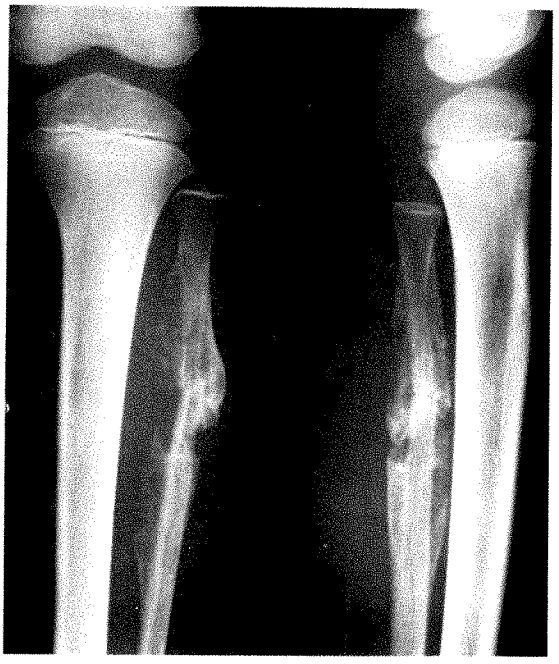


FIG. 2. The appearance of the fibular lesion at the completion of the initial course of radiation therapy. The extent of the involvement is much more clearly demarcated at this time. The "notch-like" defect along the lateral aspect represents biopsy site (October, 1956).

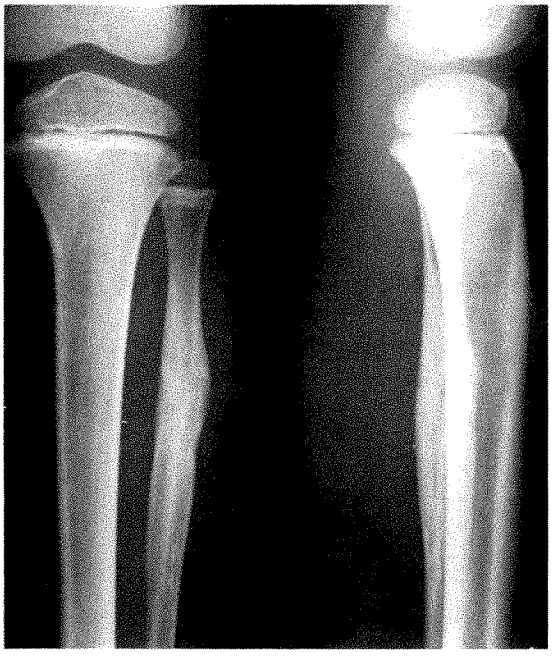


FIG. 3. The appearance of the left proximal fibula 6 months following the initial course of radiation therapy. The cortical margins are sharply defined and, except for slight widening of the fibular shaft, no abnormality is noted (March, 1957).

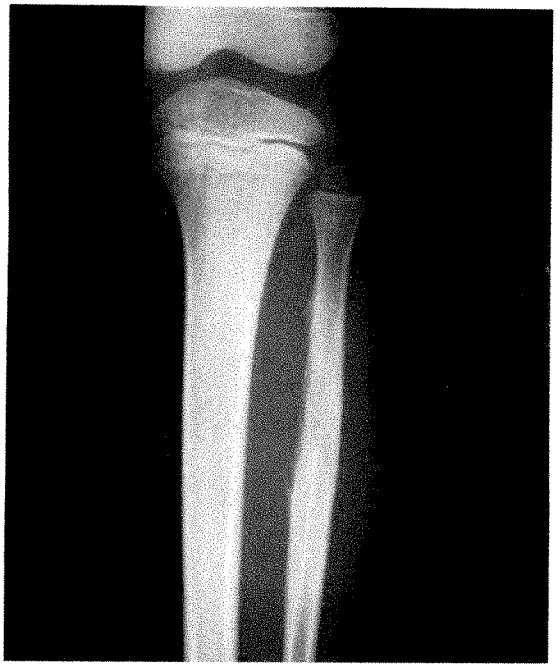


FIG. 4. Roentgenogram demonstrating evidence of recurrence (November, 1957).

a granuloma or Ewing's sarcoma. Following microscopic confirmation of Ewing's sarcoma by several experienced pathologists, including Dr. L. Lichtenstein, radiation therapy to the involved fibula was commenced on September 8, 1956 and was completed on October 8, 1956. The estimated tumor dose was 4,000 rads;

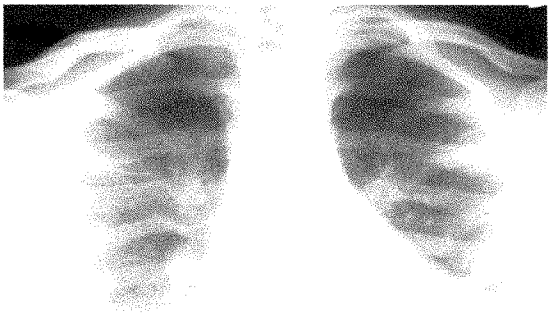


FIG. 5. Routine follow-up chest study in May, 1958 demonstrating a nodular opacity overlying the plane of the left anterior third rib.

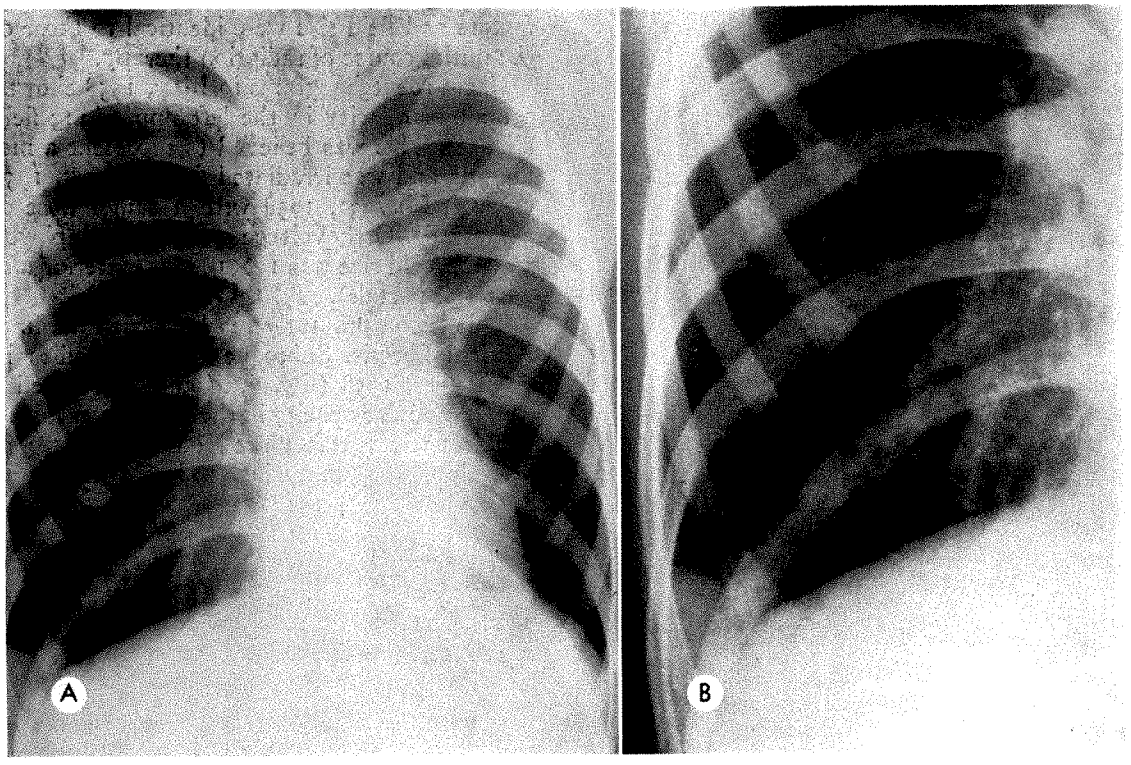


Fig. 6. (*A* and *B*) Following radiation therapy, the lesion in the left upper lung field is no longer visible. There is minimal fibrosis present (September, 1958). *A* demonstrates a nodular density at the right costophrenic angle; *B* outlines this opacity in better detail using magnification.

physical factors were 220 kv., half-value layer 1.0 mm. Cu; anterior, posterior and lateral portals measured 25×8 cm. and covered the entire shaft of the fibula except for the distal metaphysis. The appearance of the fibula at the completion of therapy is shown in Figure 2 and 3.

The patient remained well until November, 1957 when pain recurred along the lateral aspect of the proximal left lower leg. Clinical and roentgen findings at this time (Fig. 4) were interpreted as local recurrence. Following considerable discussion by the medical personnel involved, a second course of therapy was agreed

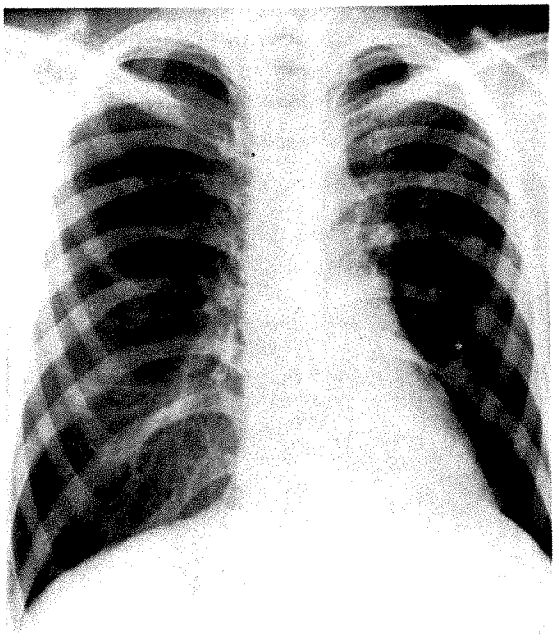


Fig. 7. The appearance of the chest in April, 1963. Other than minimal fibrosis in the left upper lung field and slight shift of the heart and mediastinum to the left, no abnormalities are noted.

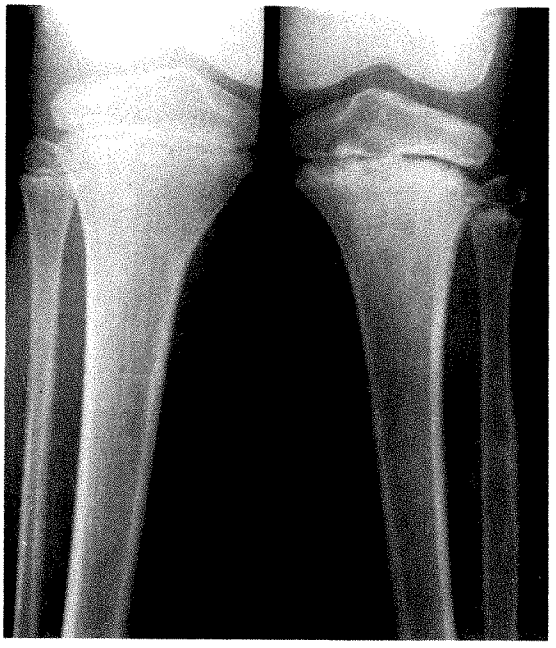


FIG. 8. A comparison of the proximal left lower leg with right (April, 1963). The fibular lesion appears well healed with no evidence of recurrence. Note the sclerosis of the metaphysis following the second course of radiation therapy and minimal valgus deformity on the left.

upon; this was started on March 4, 1957 and terminated on December 4, 1957 delivering an additional 4,000 rads to the fibula. Physical factors and field size were identical to those of the initial course of radiation therapy.

Chest roentgenograms in May, 1958 showed a nodular opacity in the left upper lung field (Fig. 5). This was believed to represent a metastasis and was irradiated from May 7, 1958 through May 31, 1958 with a tumor dose of 3,000 rads. Orthovoltage was again employed and the field size was 15×15 cm. (anterior and posterior portals).

This lesion promptly resolved with minimal residual fibrosis. Subsequent chest studies in September, 1958 (Fig. 6, *A* and *B*) showed a similar nodular opacity in the right lower lobe.

The patient was placed on large doses of penicillin, streptomycin, and chloromycetin from October 1, 1958 through January 1 of 1959. All subsequent chest studies have been normal except for the mild fibrosis in the left upper lung field (Fig. 7).

The patient has remained well, attends school and participates in numerous athletic activities. He has approximately one inch of shortening of the left leg secondary to epiphyseal arrest (Fig. 8). Figure 9, *A* and *B* shows the cellular structure of this lesion.

DISCUSSION

Many clinicians believe that the 5 year survival rate in microscopically proven Ewing's sarcoma is less than 5 per cent.

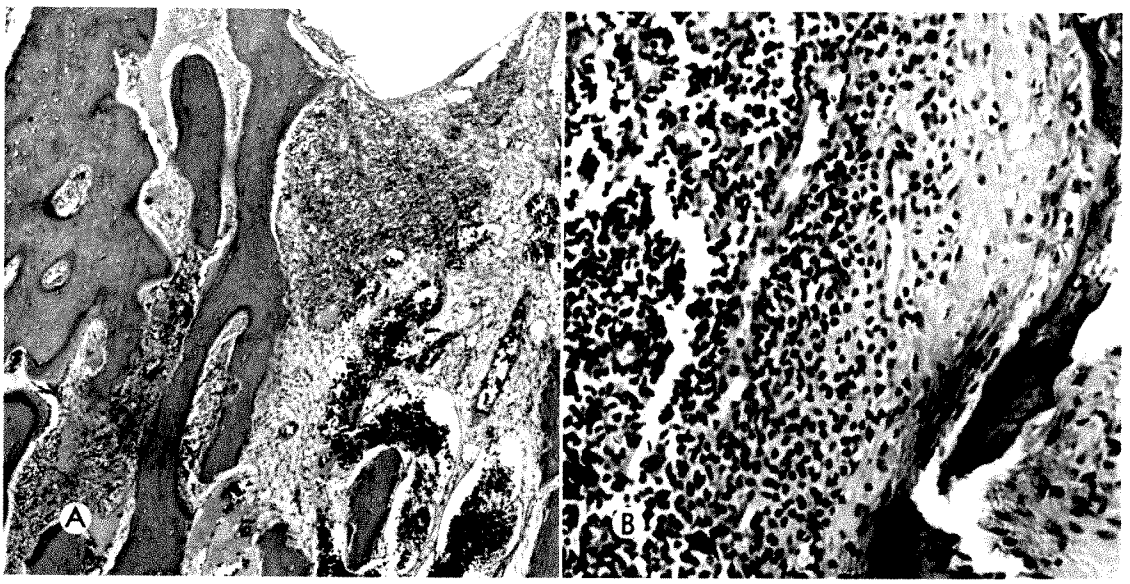


FIG. 9. (*A*) Gross low-power microscopic appearance of the biopsy specimen obtained in September, 1956. (*B*) High-power study of the cellular structure.

They are even more pessimistic regarding survival after local recurrence. Most consider pulmonary metastases a grave and probably fatal complication.

The decision to re-irradiate the local recurrence was made with the family's understanding that the possibility of amputation might eventually arise even though the neoplasm were eradicated. Epiphyseal arrest with subsequent shortening and slight angulation has been readily accepted by the parents.

While microscopic validation of the pulmonary opacities is lacking, it is contended that the roentgen appearance is virtually diagnostic of such.

For the first (left upper lobe) pulmonary metastasis to resolve following irradiation is not unusual. Whether the second (right lower lobe) represents spontaneous resolution or was, in fact, influenced by massive antibiotic therapy, is speculative but remains a possibility. A similar phenomenon was observed by one of us (RSH) in a pair of identical twins, both with Letterer-Siwe's disease. One received no treatment, the other actinomycin and penicillin. The latter had prompt resolution of both pulmonary and bony lesions. On discontinuing therapy and starting the drugs on the previously untreated child, the clinical course was reversed—bone and parenchymal disease returned in the twin now off medication, regression of the disease was promptly apparent in the other.

SUMMARY

1. A 7 year survival in a case of Ewing's sarcoma, histologically confirmed, is presented.

2. The patient had not only local recurrence 1 year following initial irradiation but pulmonary metastases 6 months subsequent to the local bone recurrence.

3. Two courses of radiation therapy controlled the primary lesion to date.

4. Radiation therapy of the left upper lobe metastasis has controlled this lesion for $5\frac{1}{2}$ years. The other pulmonary metastasis has apparently responded to vigorous antibiotic therapy.

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The authors wish to extend their sincere appreciation to Dr. Louis Lichtenstein for his help in reviewing the microscopic sections, and Drs. L. H. Garland and M. A. Sisson for their kind assistance in reviewing the manuscript.

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THE AMERICAN JOURNAL OF ROENTGENOLOGY, RADIUM THERAPY AND NUCLEAR MEDICINE

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Forty-sixth Annual Meeting: Greenbrier Hotel, White Sulphur Springs, W. Va., April 13–16, 1964.

EDITORIAL

AREAS OF CONCERN IN MAMMOGRAPHIC TECHNIQUES

ROENTGENOLOGISTS often are involved in the management of cancer of the breast and now especially so in the problems of early diagnosis. Certainly they want to contribute the full potentialities of their specialty toward an improvement of the cure rate in this important disease, a figure which has not changed much in the past decade. In this endeavor, however, they are confronted with a choice of many, often fundamentally different, roentgenographic techniques for mammography. The selection of the one best suited to the particular situation is important to the welfare of the patient.

As the use of mammography continues to grow both as a regularly employed method of physical diagnosis and in a more limited fashion as a possible survey procedure, the breasts of thousands of patients undoubtedly are being studied weekly. During this period of evaluation and expanding utilization, it is primarily important that only diagnostically satisfactory roentgenographic techniques be employed in order not to risk the health of the patient or to bring unjustified discredit upon the procedure. Particularly, caution must be exercised regarding the amount of radiation that is delivered to these glandular organs, especially in younger patients whose diagnosis of breast disease often is more difficult, requiring special, repeat or interval examinations.

Because mammography is a form of medical diagnosis in which the results expressed are a judgment based upon the examiner's training and ability, some degree of personal preference in regard to the technique selected by a radiologist can be expected. On the other hand, it must be remembered that the roentgenographic technique can and does directly affect the diagnostic value of a roentgen examination, a fact demonstrably true in mammography. In other words, the significant physical diagnostic qualities of an exposed roentgenographic film can be measured objectively with the aid of the radiophysicist. Once accurately determined, these results will limit the room for choice based on subjective criteria or personal preference.

Recent work providing such an objective evaluation apparently defined quite fully those diagnostic qualities that should be procured before a mammographic procedure can be considered medically acceptable.[1,2] It was concluded that examinations which employ either standard or non-screen film, whether used with or without screens, are seriously inferior chiefly because of the poor definition they provide. Only films of the industrial type appear to be able to produce an acceptable roentgen image. Furthermore, it was demonstrated that the faster industrial films (Type AA) produced just as good results as the slower (Type M) under the conditions imposed in roentgenography of the breast. Thus, there seems to be no valid reason for employing the latter type of film. These conclusions obviously lead the radiologist to a careful consideration of the skin and tissue doses involved in mammography.

Of all the well-established diagnostic roentgenographic procedures, mammog-

[1] SILER, W. M., GARRETT, R., SNYDER, R. E., LAUGHLIN, J. S., and SHERMAN, R. S. Mammography, an examination of techniques: (a) Exhibit and paper at Tenth International Congress of Radiology, Montreal, Canada, 1962. (b) Exhibit at Radiological Society of North America, Chicago, 1961. (c) Exhibit at American Roentgen Ray Society, Washington, D. C., 1962.

[2] SILER, W. M., SNYDER, R. E., GARRETT, R., LAUGHLIN, J. S., and SHERMAN, R. S. Development and use of mammographic technique. AM. J. ROENTGENOL., RAD. THERAPY & NUCLEAR MED., 1964, *91*, 910–916.

raphy entails the highest dose to a restricted area of the patient because of its requirements of low kilovoltage and industrial film. Low kilovoltage roentgen rays make possible the attainment of certain necessary advantages, primarily in contrast, but at the same time, they also create hazards resulting from their low energy with decreased penetration. Consequently, the relatively high skin and tissue doses required for the adequate exposure of a film become unavoidable despite an increasing response of some films to lower energy roentgen rays. This hazard is made more possible because of the rapid change in tissue absorption coefficients with small changes in kilovoltage and associated film factors in this low voltage range.

Those using industrial film of M type and actually operating their roentgenographic units at a kvp. of 26 to 30 are dealing with skin doses in the range of 10 r or more per exposure, a figure substantially higher than has been indicated in the literature dealing with this particular technique. Since a minimum of 3 exposures for each breast is recommended, a situation of concern arises. Furthermore, some radiologists, in addition to the roentgenograms in the 3 standard views, take spot, compression or special roentgenograms so that one breast may receive skin doses in excess of 50 r during the course of a single study.

It seems evident, therefore, that in mammography the radiologist has the responsibility of assuring himself by actual measurements on his own equipment as to the precise magnitude of the skin dose per exposure and preferably also of the actual kilovoltage. Neither value can be inferred from meter readings on the instrument panel nor necessarily by analogy with measurements made on equipment described in the literature.

There can be no definite dividing line with regard to that skin dose above which the radiologist has cause for concern and below which there is no contraindication. Therefore, at the present state of knowledge the radiologist is always obligated to employ the smallest amount of radiation possible to accomplish the desired result. When it has been demonstrated that two different techniques produce mammograms of comparable quality, then the one entailing definitely higher exposure to the patient should be avoided.

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NEWS ITEMS

SYMPOSIUM ON THE ASSESSMENT OF RADIOACTIVE BODY BURDENS IN MAN

The International Atomic Energy Agency (IAEA) will hold a Symposium on the Assessment of Radioactive Body Burdens in Man, in Heidelberg, Germany, May 11–16, 1964.

The Symposium will be concerned with the estimation of internal radioactive contamination in humans (either by direct methods such as *in vivo* counting or by indirect methods such as excretion analysis) and of the associated radiation dose.

The following main topics will be covered:

General Techniques: 1. Direct *in vivo* measurements of body burdens: (a) measurement of total body activity, organ activity, activity distribution; (b) processing, statistics and interpretation of data; 2. indirect methods for the assessment of body burdens: (a) sampling and sample processing; (b) measurement of samples; (c) processing, statistics and interpretation of data; and 3. analysis of the microscopic distribution of contaminants and assessment of absorbed dose.

Results and Their Interpretation: The papers to be discussed in this part of the Symposium will deal with results obtained in monitoring radium, strontium, cesium, rare earth elements, thorium, uranium, and plutonium; and with other radionuclides if relevant to the metabolism of the elements enumerated or if of particular interest to radiologic protection.

Requests for additional information should be addressed to Dr. Paul Fent, Division of Public Information, International Atomic Energy Agency, Vienna I., Kärntnerring 11, Austria.

AMERICAN MEDICAL WRITERS' ASSOCIATION

The American Medical Writer's Association—a professional society of people engaged or interested in various aspects of medical communication—will hold its Twenty-first Annual Meeting, September 24–27, 1964, in Philadelphia, the cradle of American medical education and a leading center of medical publishing.

Among the functions of the Association are the maintenance of a medical manuscript service, a roster of qualified lecturers on medical communication, the presentation of fellowships and awards for "distinguished service in medical communication" to outstanding individuals and medical publications, and the provision of scholarship assistance to students of medical journalism.

Membership is available to any person "actively engaged or interested in any aspect of communication in the medical and allied professions."

The national office is located at 2000 P Street, N.W., Washington, D. C. 20036.

COLLOQUE ANATOMO-CLINIQUE INTERNATIONAL SUR LES TUMEURS DU CORPS THYROIDE

The International Colloquium on the Malignant Tumors of the Thyroid Gland will be held June 11–14, 1964, in Marseille, France.

The subjects to be discussed are: (1) Papillary tumors of the thyroid gland, Chairman, P. Denoix, Paris; (2) anatomic clinical classification and elements of the diagnosis, Chairman, J. Delarue, Paris; (3) thyroid pathology and cancer, Chairman, T. Winship, U.S.A.; (4) general etiology of the thyroid cancers, Chairman, S. Lindsay, U.S.A.; (5) therapy of the thyroid cancers: surgery, Chairman, G. Crile, U.S.A.; and (6) therapy of the thyroid cancers: surgery and physical agents, Chairman, H. Redon, Paris.

For further information, please write to: Professeur H. Payan, Secrétaire Général, Faculté de Médecine, Boulevard d'Alès, Marseille Ve, France.

BOOK REVIEW

Books sent for review are acknowledged under: Books Received. This must be regarded as a sufficient return for the courtesy of the sender. Selections will be made for review in the interest of our readers as space permits.

CONSOLIDATED INDICES: 1958–1962. Volume VI. AMERICAN JOURNAL OF ROENTGENOLOGY, RADIUM THERAPY & NUCLEAR MEDICINE. Compiled under the direction of The Publication Committee and The Editorial Office of the American Roentgen Ray Society. Cloth. Pp. 347. Price, $23.50. Charles C Thomas, Publisher, Springfield, Ill., 1964.

Volume VI of Consolidated Indices of THE AMERICAN JOURNAL OF ROENTGENOLOGY, RADIUM THERAPY & NUCLEAR MEDICINE is issued in fulfillment of the policy of compounding every five years the author and subject references of the material published on its pages for a quick and readily accessible perusal. It is prepared with the same thoroughness as characterized the preceding five volumes, and embodies the latest and most up-to-date achievements in the rapid technical development of the graphic arts.

The volume contains approximately 25,000 items listed in alphabetical order under the name or names of the authors and the classified subject material. Both the names of the authors and the titles of the lead subjects appear in bold face type, followed by the respective subject or name entries in regular type under the same entry. The volume numbers are also set in bold arabic type and a distinction is made between the original and abstracted articles. The initial page of the originial article is prefixed with an asterisk and the initial page of the abstracted article is placed in parenthesis. Cross references are given for the names of the associated authors in case of multiple authorship and for the synonymous terms of subjects with consideration of the consistently increasing scope of the terminology in the radiologic language. This arrangement, besides simplifying the usability of the index, greatly enhances its completeness and scientific accuracy.

The late Dr. Lawrence Reynolds, illustrious editor of the JOURNAL for more than three decades, has set a high standard for the publication of these Indices. Mr. Charles C Thomas, as the Publisher, and The Banta Publishing Company, as the Printer, have combined their efforts in producing a work of distinctive quality. Volume VI is presented in the belief and hope that it will fulfill the aim of a faithful continuation of that endeavor.

T. LEUCUTIA, M.D.

BOOKS RECEIVED

MENINGIOMAS INVOLVING THE TEMPORAL BONE: CLINICAL AND PATHOLOGICAL ASPECTS. By George T. Nager, M.D., Associate Professor of Laryngology and Otology, Anatomical and Pathological Laboratories, Department of Otolaryngology, The Johns Hopkins University School of Medicine and Hospital, Baltimore, Md. Cloth. Pp. 170, with many illustrations. Price, $11.50. Charles C Thomas, Publisher, 301–327 East Lawrence Avenue, Springfield, Ill., 1963.

TREATMENT OF CANCER AND ALLIED DISEASES: TUMORS OF THE SOFT SOMATIC TISSUES AND BONE. Edited by George T. Pack, M.D., F.A.C.S., and Irving M. Ariel, M.D., F.A.C.S. Volume VIII, Second edition. Cloth. Pp. 574, with 778 illustrations. Price, $26.50. Hoeber Medical Division, Harper & Row, Publishers, 49 East 33rd Street, New York 16, N. Y., 1964.

SMOKING AND HEALTH: REPORT OF THE ADVISORY COMMITTEE TO THE SURGEON GENERAL OF THE PUBLIC HEALTH SERVICE. U. S. Department of Health, Education, and Welfare. Paper. Pp. 387, with many tables. Price, $1.25. Superintendent of Documents, U. S. Government Printing Office, Washington, D. C. 20402, 1964.

VASCULARIZATION, OXYGEN TENSION, AND RADIOCURABILITY IN CANCER OF THE CERVIX: A COLPOPHOTOGRAPHIC, POLAROGRAPHIC, AND CLINICAL STUDY. By Per Kolstad, M.D., the Gynecological Department, The Norwegian Radium Hospital; and the Norsk Hydro's Institute for Cancer Research, Oslo, Norway. Paper. Pp. 152, with many illustrations. Price, N.kr.45.-. Norwegian Universities Press, Universitetsforlaget. In the U.S.A., Box 142, Boston 13, Mass., 1963.

CLINICAL UROGRAPHY: AN ATLAS AND TEXTBOOK OF ROENTGENOLOGIC DIAGNOSIS. Second edition. By John L. Emmett, A.B., M.D., M.S. in Urology, Consultant in Urology, Mayo Clinic; Professor of Urology, Mayo Foundation, Graduate School, University of Minnesota, Rochester, Minn. Cloth. Volume II. Pp. 647 through 1294, with many illustrations. Price, $50.00 for the set. W. B. Saunders Company, Philadelphia, Pa., 1964.

Ergebnisse der medizinischen Strahlenforschung: Diagnostik, Therapie, Nuklearmedizin, Biologie, Neue Folge. Herausgeber, H. R. Schinz, Zürich; R. Glauner, Stuttgart; and A. Rüttimann, Zürich. Cloth. Pp. 561, with many illustrations and tables. Price, Ganzleinen DM 160.-. Georg Thieme Verlag, Postfach 732, Herdweg 63, 7000 Stuttgart 1, Germany. In the U.S.A. and Canada, Intercontinental Medical Book Corporation, New York 16, N. Y., 1964.

Patient Care and Special Procedures in X-Ray Technology. Second edition. By Carol Hocking Vennes, R.N., B.S., Part-time Clinical Instructor, University of Minnesota Hospital; formerly Surgical Supervisor and Clinical Instructor, University of Minnesota Hospital, Minneapolis, Minn.; and John C. Watson, R.T., Technical Consultant, Department of Radiology, Yale Medical Center and Grace-New Haven Community Hospital, New Haven, Conn.; formerly Director of Courses in X-ray Technology, University of Minnesota Hospitals, Minneapolis, Minn. Cloth. Pp. 228, with many illustrations. Price, $6.25. The C. V. Mosby Co., 3207 Washington Boulevard, St. Louis 3, Mo., 1964.

Cancer of the Stomach. By William H. ReMine, M.D., M.S. in Surgery, F.A.C.S., Head of a Section of Surgery, Mayo Clinic; Assistant Professor of Surgery, Mayo Foundation, Graduate School, University of Minnesota; James T. Priestley, M.D., M.S. in Experimental Surgery, Ph.D. in Surgery, F.A.C.S., Head of a Section of Surgery, Mayo Foundation, Graduate School, University of Minnesota; and Joseph Berkson, M.D., D.Sc., Head of the Section of Biometry and Medical Statistics, Mayo Clinic; Professor of Biometry and Medical Statistics, Mayo Foundation, Graduate School, University of Minnesota, Rochester, Minn. Cloth. Pp. 255, with many illustrations. Price, $11.50. W. B. Saunders Company, Philadelphia, Pa., 1964.

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ABSTRACTS OF RADIOLOGICAL LITERATURE

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ROENTGEN DIAGNOSIS

Blood and Lymph System

DeBakey, Michael E. Basic concepts of therapy in arterial disease. (Lasker Award.) *J.A.M.A.*, Nov. 2, 1963, *186*, 484–498. (From: Baylor University College of Medicine, Texas Medical Center, Houston, Texas.)

Great progress has been made in recent years toward better understanding of the insidious pathologic anatomic changes that occur in the arterial wall, more precise methods of diagnosis, and more effective therapy.

The concept has evolved that, regardless of etiology, the lesion in many forms of aortic and arterial disease may be well localized and is segmental in nature with a relatively normal, patent proximal and distal arterial bed.

It becomes possible to classify most arterial diseases into two major types: occlusive lesions and aneurysms. In both types the lesions may be of congenital, acquired, or traumatic origin. In terms of the surgical approach to the problem, the nature, location and extent of the lesion itself or the resultant hemodynamic disturbances assume greater significance than these etiologic factors. Thus, an aneurysm of the aorta may be of arteriosclerotic, traumatic, syphilitic or dissecting origin. The most common etiologic factor is arteriosclerosis or atherosclerosis. Combined forms of these lesions often occur in the same patient.

Occlusive lesions. The *first category* of arterial occlusive lesions is concerned with lesions involving the major branches of the aortic arch. Two characteristic patterns may be recognized, namely, a proximal form, involving the major arteries arising from the aortic arch, and a distal form, involving the internal carotid and vertebral arteries at their respective origins. The occlusive process may be complete or incomplete. Multiple lesions are present in over half the cases.

On the basis of the author's observations, a concept has developed of the totality of cerebral blood flow. The brain receives its arterial blood supply from for major arterial systems; namely, the two vertebral arteries and the two internal carotid arteries. Both extracranially and intracranially these systems may communicate with each other through major collateral channels. Ischemic disturbances may then become apparent only when the collateral circulation is inadequate to meet the demands of the tissues it supplies.

Still another important conceptual consideration is the distinction between tissue viability and normal function. It has long been known that certain degrees of ischemia may inhibit normal function but maintain viability of the tissues even for prolonged periods.

Involvement of the visceral branches of the abdominal aorta, namely the celiac axis, superior mesenteric and renal arteries, comprises the *second category* of arterial occlusive disease. Etiologically, the occlusive process in this group is atherosclerosis in the great majority of cases. The ischemic disturbances resulting from this category of occlusive disease produce two characteristic, but widely different, clinical entities, namely, abdominal or intestinal angina and renovascular hypertension.

The *third category* of occlusive disease involves the terminal abdominal aorta and its major branches and tends to assume two recognizable patterns permitting a similar classification into two subgroups: a proximal form involving the aortoiliac region and a distal form involving the femoropopliteal arteries.

The *final category* of occlusive disease, and perhaps the most commonly lethal, involves the *coronary arteries*. The predominant underlying lesion is arteriosclerosis or atherosclerosis. Surgical therapy is applicable to such occlusive lesions of the coronary arteries, if segmental in nature with a patent proximal and distal arterial bed.

The primary objective of therapy is correction of these disturbances by restoration of normal circulation. Principles of surgical therapy designed for this purpose have been developed and consist essentially of thromboendarterectomy, excision with graft replacement, the bypass graft, and patch graft angioplasty.

A most important consideration is the development of a satisfactory and effective arterial substitute. The flexible, seamless, knitted dacron tube was found to meet the essential requirements of strength, durability, and function. Experience with this graft replacement now includes over 5,000 cases and extends over a period of 8 years. Successful results have been achieved in from 80 to 97 per cent of the cases. In approximately 900 cases of occlusive disease which produced cerebrovascular insufficiency, circulation was restored in 92 per cent. Relief of symptoms has been maintained in approximately 90 per cent of patients who had transient cerebral ischemia, 95 per cent of those with progressive stroke, and 70 per cent of those with completed stroke. In approximately 300 cases of renal artery occlusive disease associated with hypertension, successful results with relief of hypertension were obtained in 80 per cent. In approximately 3,300 patients with arterial insufficiency of the lower extremities, successful results were obtained in 93 per cent.

Aneurysms. Emphasis has shifted from etiology to nature and location of the aneurysmal process. Thus extirpation, or complete obliteration, of the lesion is essential to therapy. Fusiform aneurysms, for example, are usually treated by excising the diseased segment and replacing it with a dacron graft.

Aneurysms lend themselves to classification as follows: (1) In the aortic arch, (2) in the descending thoracic aorta, (3) in the thoracoabdominal segment

of aorta, and (4) in the abdominal aorta distal to the renal arteries.

The results and the operative risk are greatly influenced by a number of factors, including particularly the type and location of the lesion, age of the patient, acute rupture, pre-existing heart disease, and other serious associated disease.

It now appears from these long-term arteriographic studies that extend up to 10 years that the lesions not only may remain localized for long periods of time, but also may stop progressing once the lesion has reached a certain stage.—*Stephen M. Tager, M.D.*

HANAFEE, WILLIAM. Axillary artery approach to carotid, vertebral, abdominal aorta, and coronary angiography. *Radiology*, Oct., 1963, *81*, 559–567. (From: Department of Radiology, U.C.L.A. Center for the Health Sciences, Los Angles, Calif.)

Since August, 1961 the transaxillary approach has been used in approximately 200 vascular examinations at the U.C.L.A. Medical Center. Severe arteriosclerotic involvement of the iliac arteries makes transfemoral catheterization technically difficult and sometimes prohibitively dangerous. Because of this an alternative approach for visualization of the carotid vertebral and coronary arteries is desirable and the author feels that the axillary artery is a convenient vessel to use due to its accessibility and its relatively fixed position on the teres major tendon high in the axilla.

The equipment and techniques used are described in detail. The guide wires and catheters required some modification over the conventional Seldinger techniques. The size and type of catheter used depend on the examination to be performed.

Only 4 complications occurred in this limited series and 3 of these occurred in the first 7 cases while the technique was being worked out. The first complication was a temporary hemiparesis and the next one was a large local hematoma. In the third case some transient pains developed, radiating to the third, fourth and fifth fingers. The most recent complication was some vascular embarrassment of the upper extremity when the catheter totally occluded the subclavian artery at its take-off from the aortic arch.

The author feels that axillary artery catheterization is the method of choice in vertebral angiography and coronary angiography and is an alternate method of examination of the abdominal aorta and renal arteries in poor risk patients with iliac occlusive disease. The radiologist who familiarizes himself with the axillary approach to catheter angiography is in a position to assist his colleagues in a wide variety of special studies.—*Donald N. Dysart, M.D.*

MARSHALL, THOS. R., LING, J. T., and GONZALEZ, RAUL. Additional experiences with direct percutaneous noncatheter brachial angiography—left panarteriography—right cerebral angiography. *Radiology*, Oct., 1963, *81*, 568–575. (From: Department of Radiology, University of Louisville School of Medicine, Louisville, Ky.)

A new method of direct percutaneous noncatheter brachial angiography is described with which the authors have performed 175 examinations for a total of 285 injections without fatality or serious morbidity. Right brachial studies were made in 85 patients, and left brachial in 90.

After appropriate premedication, brachial artery puncture is performed, using a special 17-gauge vascular needle, at a level with a line 1 cm. above the epicondylar processes of the humerus. For right cerebral angiography 25 to 30 cc. (adults) of meglumine iothalamate 60 per cent is used and for left panarteriography studies, 30 to 50 cc. (adults) of sodium iothalamate 66.8 per cent or 50 to 70 cc. of meglumine iothalamate 60 per cent. A pressure injector must be employed and a pressure cuff should be placed around the arm distal to the site of injection and inflated above the systolic pressure before injection.

Right brachial injection provides complete visualization of the right carotid-cerebral and vertebral-basilar systems. When pressure is applied to the left common carotid artery during right brachial injection, the complete cerebral and basilar arterial system can be demonstrated.

Left brachial injection gives excellent opacification of the thoracic and abdominal aorta and the renal, iliac and femoral arteries. Renal arterial studies have shown the same or better contrast than from percutaneous transfemoral renal arteriography.

For demonstration and staging of bladder tumors according to Jewett's hypothesis, 50 to 75 cc. of carbon dioxide is instilled into the bladder prior to left brachial artery injection and pneumatic tourniquets are used to occlude both femoral arteries. This gives good visualization of the vessels supplying the wall of the bladder.

Direct noncatheter brachial angiography, as described, avoids the hazards inherent in such other approaches as direct needle puncture of the carotid or vertebral artery, percutaneous transfemoral catheterization by the Seldinger technique, and translumbar aortography.—*Ralph Bowman, M.D.*

AMPLATZ, KURT, RESCH, JOSEPH, and HILAL, SADEK. A catheter approach for cerebral angiography. *Radiology*, Oct., 1963, *81*, 576–583. (From: Department of Radiology, University of Minnesota Hospital, Minneapolis, Minn.)

This method was developed for evaluation of a group of patients with stroke syndrome in the athero-

sclerotic age group. Since direct needle puncture of diseased arteries is associated with a higher incidence of local complications, an indirect approach was used by catheterization of a single peripheral artery. By using the right subclavian artery a specially shaped short catheter may be employed, permitting good manual control and therefore selective catheterization of the right vertebral artery, of the right carotid, the left carotid and left sublcavian artery. The equipment and technique are described in detail.

Forty-three patients with well established stroke syndrome were examined by this procedure. Age range was from 29 to 81 years with 75 per cent of the patients more than 60 years old. Ten of the 43 patients with clinical signs and symptoms of stroke had angiographically normal arteries as determined by the complete 4-vessel study. Complete carotid obstruction was present unilaterally in 4 and bilaterally in 2. Pathologic conditions were found also in many of the vessels visualized. Generally, however, the clinical picture did not correlate with the severity of the vascular disease. Blood flow and pressure measurements were also carried out during the arterial catheterization in order to evaluate the hemodynamic significance of demonstrated obstructing lesions.

Complications consisted of a mediastinal hematoma with spontaneous regression, a grand mal seizure followed by transient blindness and aphasia, and a local hematoma requiring no special treatment. Three patients had transient blindness which subsided with conservative measures. It was felt that the episodes of transient blindness were not due to the selective injection of contrast medium in the vertebral arteries but to a systemic hypotension resulting in a decreased cerebral perfusion.

One of the main purposes of this project was the evaluation of the natural history of occlusive atherosclerotic disease in patients with clinical evidence of cerebral vascular insufficiency. A complete angiographic and clinical re-evaluation is contemplated at yearly intervals in order to determine the progress of obstructing disease.—*Donald N. Dysart, M.D.*

Glenn, Frank, Evans, John A., Halpern, Mordecai, and Thorbjarnarson, Bjorn. Selective celiac and superior mesenteric arteriography. *Surg., Gynec. & Obst.*, Jan., 1964, *118*, 93–100. (From: Departments of Surgery and Radiology, The New York Hospital-Cornell Medical Center, New, York, N. Y.)

By the Seldinger percutaneous technique, a catheter is introduced into a femoral artery and its tip placed into the celiac artery. A second catheter is inserted into the opposite femoral artery and its tip placed into the superior mesenteric artery. Contrast material is injected simultaneously into both vessels and films are exposed in various positions. It is important to be familiar with the normal variations of the vessels. Abnormalities to be sought are displacement, narrowing, and occlusion of vessels, abnormal collaterals, and areas of increased or decreased vascularity.

Three case reports are presented in detail. One patient showed stretching of the superior pancreaticoduodenal artery and its branches as well as of the pancreatic branches of the gastroduodenal artery secondary to a carcinoma in the head of the pancreas. A second patient revealed displacement of the superior mesenteric, hepatic, gastroduodenal and superior pancreatico-duodenal arteries due to a large cystadenoma of the pancreas which also showed a tumor stain. A third patient had known metastatic gastric carcinoma and developed jaundice; a narrowed hepatic artery indicated the presence of a mass in the hilus of the liver which was confirmed at autopsy.

Over 40 celiac and superior mesenteric arteriographies have been done without complications. The procedure is recommended as an additional method in the diagnosis of frequently obscure lesions of the abdominal viscera, especially the liver and pancreas.—*Steven H. Cornell, M.D.*

Gütgemann, A., Schreiber, H. W., and Esser, G. Unusual method of portacaval anastomosis: anastomosis between the left gastric vein and the inferior vena cava. *German Med. Monthly*, Aug., 1963, *8*, 331–335. (From: Department of Surgery, University of Bonn, Germany.)

The authors discuss portal hypertension and its surgical management. In cases of generalized portal hypertension, usually due to cirrhosis, they recommend a portacaval anastomosis using the most direct method possible. In cases of prehepatic block with congenital or acquired intra- or extrahepatic vascular stenosis, they advise splenectomy and usually a splenorenal anastomosis.

Some cases cannot be treated in this manner due to underdevelopment, malformation, or other factors making the mobilization and usual anastomosis technically difficult. Occasionally, these cases can be handled by an anastomosis between the left gastric vein and the inferior vena cava. This produces a decompression shunt. It is relatively simple from a technical standpoint, and may be of considerable practical value in selected cases. *George L. Sackett, Jr., M.D.*

RADIATION THERAPY

Howard, Norman. Phlebography in superior vena caval obstruction. *Radiology*, Sept., 1963, *81*, 380–384. (From: Radiotherapy De-

partment, Royal Marsden Hospital, London, England.)

Phlebography is recommended as a valuable, simple, safe diagnostic procedure for most patients suffering from superior vena caval obstruction. This conclusion is based on a study of 54 cases of superior vena caval obstruction treated at the Royal Marsden Hospital, London, England, during the last 3 years. Phlebography was carried out to elucidate the hemodynamics in this condition and to correlate the findings with treatment and prognosis. Their procedure is relatively simple. Both basilar veins are utilized for simultaneous injection of 20 to 30 cc. of 45 per cent hypaque solution. Four films are exposed at one-second intervals.

Phlebography furnished valuable information not obtained by other means. The site and extent of the obstruction and the amount of collateral circulation could be demonstrated. Involvement of the wall of the vein by tumor could be seen as a constant patchy filling defect.

Information gained from phlebography was found to be helpful in deciding the question of anticoagulant therapy, and in determining irradiation techniques. Patients with well developed collateral circulation were found to require comparatively small radiation doses for good clinical response and return to a state of compensation. With poor collateral circulation, comparatively high doses were necessary to produce clinical relief. With radiation doses above 3,000 r, recurrence of the obstruction usually did not occur.—*A. W. Sommer, M.D.*

RUBIN, P., GREEN, J., HOLZWASSER, G., and GERLE, R. Superior vena caval syndrome: slow low-dose versus rapid high-dose schedules. *Radiology*, Sept., 1963, *81*, 388–401. (From: Strong Memorial Hospital, Rochester, N. Y.)

Acute superior vena caval obstruction is one of the true emergencies encountered in radiation therapy. The clinical literature is somewhat inconclusive as to treatment, in most cases small doses of radiation and/or chemotherapy being advocated. The authors have instituted a relatively high rapid-dose schedule which has been successful, and they have modified accordingly their clinical approach to the problem.

Generally, the diagnosis is made from this history and physical examination, plus a chest roentgenogram showing a mediastinal mass. Confirmatory studies include infrared photography, elevated venous pressure determination, occasionally phlebography, and if feasible, microscopic examination. Treatment is instituted without waiting for pathologic diagnosis, however. Central nervous system involvement is frequent. Particularly important is the electrolyte imbalance (water retention) with associated edema.

A review of 13 patients with the low-dose schedule advocated by many authors (50–100 r/day tumor dose) showed occasional worsening before improvement, and slow regression of symptoms. Seven patients have been treated with a high-dose schedule (average 400 r/day). The technique has varied, but usually supervoltage was employed with a maximum dose of 4,000 r in 10 days. Treatment is started using large fields, with smaller doses and fields employed as improvement occurs. The high-dose schedules seemed uniformly more successful in producing earlier relief and maximum improvement sooner than low-dose schedules.

Auxiliary measures may include 8 per cent urea in invert sugar given by vein; diuril and other diuretics may be administered concurrently. Chemotherapy is generally not used concurrently but might be indicated if only orthovoltage irradiation is available.—*J. C. Moore. M.D.*

HOLMES, K. S. The treatment of superior vena caval syndrome by high-dose grid technic. *Radiology*, Sept., 1963, *81*, 402–405. (From: Christie Hospital and Holt Radium Institute, Manchester, England.)

The author describes his method of treatment of superior vena caval syndrome due to carcinoma of the bronchus. One hundred thirty cases were analyzed. A dose of 4,000 to 5,000 rads was given to one anterior mediastinal field. In most cases 300 kv. roentgen rays were used and the field size varied. A 40 per cent transmission lead grid was employed in all cases and the entire dose was administered in one session.

Response was considered to be excellent in 48 per cent of the cases and some palliation was achieved in an additional 33 per cent.

Complications attributable to the radiation were few. The short treatment period is considered an advantage for patients who are expected to survive for only a few months.—*McClure Wilson, M.D.*

CHU, FLORENCE C. H., NISCE, LOURDES, and LAUGHLIN, JOHN S. Treatment of breast cancer with high-energy electrons produced by 24-mev. betatron. *Radiology*, Nov., 1963, *81*, 871–880. (From: Departments of Radiation Therapy and Physics, Memorial Hospital for Cancer and Allied Diseases, James Ewing Hospital, and the Sloan-Kettering Institute for Cancer Research, New York, N. Y.)

This paper reports further experience with electron beam therapy in carcinoma of the breast. Of the 218 patients treated, 50 were for inoperable carcinoma, and 135 for metastases; the remaining 33 received postoperative irradiation. The electron beam, with the physical characteristics of abrupt termination of radiation, controllable depth of penetration,

and no differential bone absorption, was considered desirable for further evaluation as being superior to conventional methods. The electron stream originated in a 24 mev. betatron and the energies used varied from 6 to 22 mev., depending on the desired depth of penetration; polystyrene wedges were employed occasionally.

The treatment was planned to deliver approximately 6,000 rads to the breast in 6 to 7 weeks, and 4,000–5,000 rads to the lymph nodes in 4 to 5 weeks. The skin dose was 80 to 90 per cent of the incident dose. Recurrent or metastatic tumors received 3,000 to 5,000 rads in 3 to 5 weeks. Conversion factors for computation of doses are described.

Clinical evaluation of tumor response showed a favorable response in 39 of the 50 patients treated for inoperable carcinoma, with a mean duration of regression of tumor of 12 months. One hundred of 135 patients with recurrent or metastatic disease showed improvement with a mean duration of improvement of 8 months. Skin erythema became noticeable at 1,500 to 2,000 rads; when a skin dose of 4,000 to 4,500 rads was given in 4 to 5 weeks, there was moderate to intense erythema, usually followed by dry peeling with minimal tanning. A dose of 6,000 rads to the entire breast usually produced a brisk reaction, frequently followed by moist desquamation which cleared without sequelae. Radiation pneumonitis and fibrosis had an incidence of 8 per cent and retrospective analysis suggests the causes for this. One patient had spontaneous fractures of several ribs, most likely due to hot spots caused by misalignment of adjacent fields. Systemic reactions were minimal.

The incidence of fibrosis and pneumonitis was lower with betatron therapy than with supervoltage and much lower than with orthovoltage. Very large breasts may be better treated with supervoltage, or tangential fields requiring more than 16 cm. separation. However, the authors feel that the advantages of electron beam therapy for superficial tumors such as breast outweigh the disadvantages, and have found the method to be worthwhile, convenient, and effective.—*J. C. Moore, M.D.*

Lawrence, John H., Tobias, Cornelius, A., Born, James L., Gottschalk, Alexander, Linfoot, John A., and Kling, Robert P. Alpha particle and proton beams in therapy. *J.A.M.A.*, Oct. 19, 1963, *186*, 236–245. (From: The Donner Laboratory and Lawrence Radiation Laboratory, University of California, Berkeley, Calif.)

Heavy particles are so called because they actually are heavy when compared with the weight of the electron. The neutron is approximately 1,800 times the weight of the electron and other particles are proportionately heavier according to the number of protons and neutrons within their nuclei. These particles have distinctive physical characteristics. Irradiation with heavy particles produces dense ionization in tissue and such a beam has great penetration with little scatter. Moreover, the denser ionization of the tissue at the Bragg peak produces a greater RBE and irradiation with heavy particles has been observed to be relatively independent of the oxygen effect over a portion of the Bragg peak.

With the possibility of irradiating localized lesions in tissues with little damage to surrounding structures, uses for these particles in neoplastic, metabolic and neurologic diseases have been found. The paper summarizes a 10 year experience with this form of therapy.

HYPOPHYSEAL ABLATION. Treatment to the pituitary gland was carried out in patients with breast cancer, diabetic retinopathy, acromegaly, Cushing's disease and malignant exophthalmos.

Breast cancer. A total of 159 patients with advanced metastatic disease was treated to the pituitary gland with 900 mev. alpha particles in doses of 14,000–17,000 rads in 11 days. Complete endocrine evaluations were done on all of the patients before and after treatment. Remission of disease occurred in about 30 per cent of cases with those patients who had metastasis to bone showing a better response than those with soft tissue and organ metastasis. Results approximative of those of surgical hypophysectomy were obtained.

Diabetic retinopathy. On the basis that diabetic retinopathy disappeared in patients who developed spontaneous hypopituitarism a total of 79 patients with severe diabetic retinopathy received pituitary irradiation, 8,000 to 12,000 rads, in an attempt to slow down the progressive disease process. Subjective visual improvement as well as objective improvement of the eye grounds did appear in some patients but in general the retinopathy was seldom significantly improved.

Acromegaly. Twenty-one patients received 3,000–7,000 rads in 11 days to the pituitary gland with heavy particle beams. Both subjective and objective improvement occurred in these patients. Of note is the fact that 6 of 9 patients with abnormal glucose tolerance curves prior to treatment showed return to normal of these curves after treatment. The authors believe that this represents the first incidence of externally delivered energy which influenced the metabolic function of the acromegalic pituitary gland.

Cushing's disease. Three patients were treated and all had favorable remissions of some degree with doses from 5,000–10,000 rads to the pituitary gland.

DIRECT TUMOR THERAPY. Heavy particle irradiation was carried out in visceral neoplasms and brain tumors. In the treatment of patients with brain tumors, 1 with recurrent oligodendroglioma and 2 others with astrocytomas, the ability of heavy particle beams to sterilize the treated field was demonstrated. Recurrences in 2 of these 3 patients were outside the treatment field.

Heavy particle, high energy beams are also being used in other conditions such as Parkinson's disease, intractable pain and psychic disorders, with effective results.—*Philip Rubin, M.D.*

Delahaye, R. P., Isnard, J., and Pyard, M. Les lymphosarcomes du caecum. (Lymphosarcoma of the cecum.) *J. de radiol., d'électrol. et de méd. nucléaire*, Oct., 1963, *44*, 625–634. (From: Services d'Électroradiologie et de Chirurgie de l'Hôpital militaire d'Instruction Dominique-Larrey, à Versailles [Seine-et-Oise], France.)

Lymphosarcoma of the digestive tract is rather infrequent and involvement of the cecum is very rare, there being less than 100 cases reported in the literature. One of the difficulties encountered in surveying the literature is the fact that English language publications use the term "malignant lymphoma" to include lymphosarcoma and other conditions; therefore, a satisfactory statistical evaluation of the incidence of the disease cannot be easily obtained.

Lymphosarcoma of the cecum may occur at any age between 3 and 73 years. The highest incidence, however, is in the first two decades of life. The condition is more prevalent in men, being 4 times more frequent than in women.

From the clinical point of view lymphosarcoma of the cecum often constitutes a surgical problem. The patient presents symptoms of abdominal pain, usually right lower quadrant pain, difficulty in transit of the intestinal contents with partial and eventual complete obstruction, marked and early inanition and a clinically palpable tumor.

The lesion develops in the submucous lymphatic structures of the cecum often involving the ileocecal region as well as the distal ileum. The growth of the tumor may be longitudinal involving a large segment of bowel or it may be circular with intrusion into, and narrowing and eventually occlusion of, the intestinal lumen. Erosion of the mucosa may occur, resulting in bleeding.

The authors report 2 cases, both males of 23 and 34 years of age respectively. The presenting symptoms were right lower abdominal pain, palpable right lower abdominal mass, loss of weight and incomplete obstruction.

The importance of contrast studies of the gastrointestinal tract in the diagnosis of this lesion as well as in determining its extent and location is stressed.

Treatment may be by means of (1) surgery, (2) radiation therapy, and (3) chemotherapy. In the authors' experience the first two, singly or in combination, are the recommended methods of treatment.

Five figures representing reproductions of roentgenograms and sketches and 1 table accompany this article.—*William H. Shehadi, M.D.*

Chiappa, S., Galli, G., Barbaini, S., Ravasi, G., and Bagliani, G. La lymphographie peropératoire dans les tumeurs du testicule. (Preoperative lymphangiography in testicular tumors.) *J. de radiol., d'électrol. et de méd. nucléaire*, Oct., 1963, *44*, 613–624. (From: Institut de Radiologie de l'Université de Milan, and Institut National Victor-Emmanuel-III pour l'Étude et le Traitement des Tumeurs, Milan, Italy.)

The authors report their experience with lymphangiography in 9 cases of testicular tumors. Instead of performing lymphangiography by introducing the contrast material into the web between the toes of the foot, as described by Kinmonth, the authors used the collecting lymphatic trunks of the spermatic cord.

After surgical removal of the involved testicle approximately 5 cc. of "ultrafluid" lipiodol was injected into several lymphatic trunks of the spermatic cord. The injection must be done slowly, introducing approximately 1 cc. in 8–10 minutes, so that slipping of the needle or rupture of the lymphatic channels is avoided. In 2 cases in which lymphangiography was performed, both through the spermatic cord and the webs of the toes, it was found that the lymph nodes were more adequately opacified through the spermatic cord.

This technique developed and reported for the first time by the authors is recommended as a routine procedure following orchidectomy so as to clearly delineate the draining lymphatics and identify the lymph nodes which may be enlarged due to metastatic malignant disease. Identification and localization of the involved lymph nodes in this manner is of utmost importance in determining the fields to be used in radiation therapy. The contrast material is retained in the lymph nodes for a sufficient length of time to permit follow-up examination after radiation therapy. The large lymph nodes are seen to be reduced in size or an abnormal pattern to return to normal. The displaced lymphatic channels due to enlargement of the retroperitoneal lymph nodes return to normal position, indicating successful results from radiation therapy.

There are no contraindications and no ill effects have resulted from this examination.—*William H. Shehadi, M.D.*

RADIOISOTOPES

Seitzman, David M., Halaby, Fouad A., Flanagan, Paul, Wright, Robert, and Freeman, James H. Intralymphatic radioisotope therapy. *Surg., Gynec. & Obst.*, Jan., 1964, *118*, 52–58. (From: Urology Service, Departments of Surgery, Radiology, and Pathology, Walter Reed General Hospital, Washington, D. C.)

The authors propose to treat neoplasms which are primary in, or which have extended to, the abdominal and pelvic lymphatics and lymph nodes by the intralymphatic injection of ethiodol tagged with I^{131}. This concept is not a new one, for over the years investigators have injected radioactive colloids and cytotoxic drugs in the hope that these agents might reach lymph nodes and destroy tumors there. Inevitably the problem is the same as that which confronts the present authors in that lymphatics blocked by tumor will not permit the passage of the therapeutic agent, and lymph nodes occupied by tumor will not take up tumor-lethal quantities of the agent. The proposed method must then find its applicability in those cases in which tiny foci of lymphomatous disease or small lymph node metastases are present.

The authors have employed their technique in a study of 15 patients who had, in the main, testicular carcinomas, and who were to undergo lymphadenectomy. Total dose, delivered in the form of I^{131} substituted for the inert iodine which makes up 37 per cent of the weight of the ethiodol molecule, varied from 3.5 to 33.03 millicuries. Dose calculations were attempted on the basis of the preoperative lymphangiogram, and an attempt was made postoperatively to relate the calculated dose to the apparent radioactive content of the lymph nodes from the operative specimen.

The study is an interesting one, but the method described seems to have a limited range of application to malignant disease involving lymph nodes.—*Frederick J. Bonte, M.D.*

Croll, Millard N., Shuman, Larry H., Stanton, Leonard, and Czarnecki, Casimir. Radioisotope placentography: a bedside procedure. *J. Nuclear Med.*, Nov., 1963, *4*, 417–425. (From: Departments of Radiology and Obstetrics and Gynecology, Hahnemann Medical College, Philadelphia, Pa.)

The authors present a unique method of determining the position of the placenta by the use of I^{131} labeled human serum albumin. The attempt was to develop a reliable, safe, and convenient method, and one that could be applied at the patient's bedside.

The method consists of giving 20 drops of Lugol's solution orally to the patient 2 hours prior to the study. Five microcuries of radioiodinated human serum is then injected intravenously and 10 minutes allowed for distribution of the material in the maternal circulation. The abdomen is divided into 13 counting areas with the xiphoid process acting as a reference point. Counts are made over these areas with a miniaturized scintillation detector and with the use of a small portable decade scaler. Specific details are given as to the placing of the detector. The counting areas are illustrated.

Fifty-seven patients were studied. The diagnostic accuracy of localization of the placenta was 89.5 per cent. Confirmation of the results was obtained by cesarean section or manual extraction of the placenta at the time of delivery. Five figures are included to illustrate the results of various positions of the placenta.

There follows a discussion of the radiation hazards of the procedure. Whole body radiation to the fetus was estimated at 6.5 mrads and to the mother, 15.0 mrads. The dose to the fetal thyroid was estimated at 2,500 mrads and 250 mrads to the mother's thyroid. These thyroid doses are without blocking with Lugol's solution; they are compared with 1,000 mrads to the whole body and thyroid of the fetus and 5 mrads to the maternal thyroid which are obtained during exposure of 2 abdominal scout roentgenograms.—*William K. Littman, M.D.*

Hine, Gerald J. Color-coded digital print-out for radioisotope scanning. *J. Nuclear Med.*, Nov., 1963, *4*, 439–445. (From: Radioisotope Service, Boston Veterans Administration Hospital; Department of Medicine, Boston University School of Medicine; and the Boston University Medical Center, Boston, Mass.)

Numerical print-outs are helpful for locating areas of increased activity in radioisotope scanning, but are not practical for outlining an organ or the isotope distribution within an organ. Black and white print-out systems delineate boundaries between areas of high and low concentrations very well, but may obscure a gradual change in concentration or miss an area of low concentration due to background cut-off selection.

Quantitative information can be obtained from a scan if the color of the print-out changes with the counting rate. This allows the printing pattern to show isotope distribution and the printing color indicates the variations in concentration.

Eight parallel colored typewriter ribbons are employed. A special carriage device that moves the colored ribbons beneath the printing stylus is controlled by a count-rate meter so that the printing color changes with the count rate. The ribbons are arranged in a rainbow spectrum with red on one end and purple on the other. First an attempt is made to locate the highest count rate in the organ to be scanned. The rate meter is then adjusted to about an 88 per cent level of the counting rate so that red is printed at this level down to about the 13 per cent level where purple is printed. Seldom is there difficulty in the choice of these two selections.

Four figures of color-coded print-out scans are presented: one thyroid, one liver, and two kidney.

A change in color delineates clearly areas of different count-rate without losing any information due to a preselected cut-off level. This provides an unambiguous interpretation of the information contained in a radioisotope scan.—*William K. Littman, M.D.*

MISCELLANEOUS

Schüssler, Richard. Untersuchungen über die Stabilität jodhaltiger, wasserlöslicher Röntgenkontrastmittel unter der Einwirkung von sichtbarem Licht, Ultraviolett und radioaktiver Strahlung. (Investigations of the stability of water-soluble, iodinated, roentgen contrast media to exposure to visible and ultraviolet light and to radiation from radioactive material.) *Strahlentherapie*, Oct., 1963, *122*, 311–319. (From: Strahleninstitut der Universität des Saarlandes, Homburg/Saar, Germany.)

The stability of the iodine-binding of the contrast media, abrodil, per-abrodil, urografin, and biligrafin, to exposure to visible light, ultraviolet light, and the radiation from I^{131} was investigated. The following conclusions were reached:

(1) All the contrast media investigated lost iodine from their molecules under the influence of these radiations.

(2) The aromatic compounds (per-abrodil, urografin, and biligrafin) yielded the iodine as iodide ion, but at concentrations of less than 10 per cent free iodine also appeared.

(3) The aliphatic compound (abrodil) yielded mainly free iodine.

(4) Simply using light-tight storage for contrast media, and low specific activity in tagged substances, prevented measureable separation of iodine from the contrast molecule.—*Henry G. Moehring, M.D.*

Lyon, Harvey W., Conard, Robert A., and Glassford, Kenneth F. Long-term intra-oral findings in humans after exposure to total-body irradiation from sudden radioactive fallout. I. Five year postdetonation studies. *J. Am. Dent. A.*, Jan., 1964, *68*, 31–38. (Address: Dr. H. W. Lyon, Bethesda, Md.)

The study of 239 Marshallese exposed to total body irradiation in February, 1954, from fallout after detonation of a 15 megaton thermonuclear device is presented with special reference to long term intra-oral findings.

The authors conclude that the incidence of caries and periodontal disease was high in both irradiated and nonirradiated control subjects. There appeared to be no significant increase in diseases of oral structures directly or indirectly attributable to total body exposure to doses of from 15 to 175 roentgens.—*David C. Alftine, M.D.*

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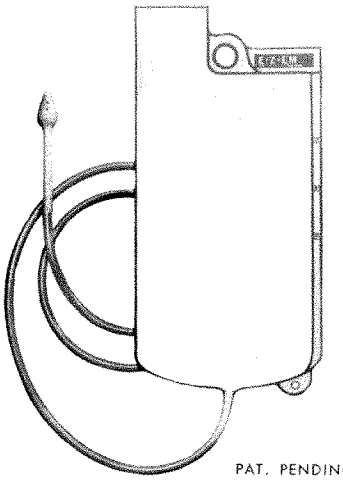
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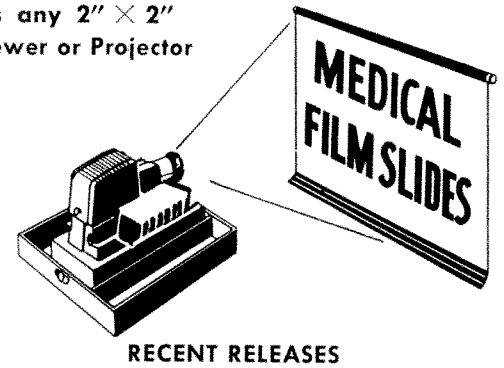
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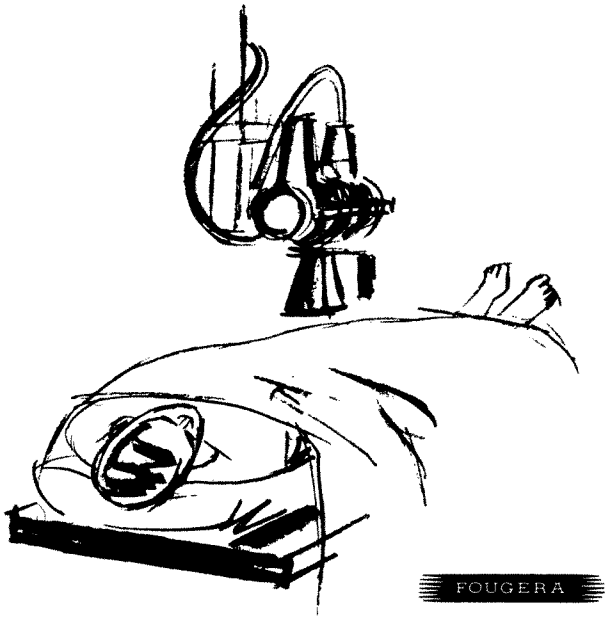
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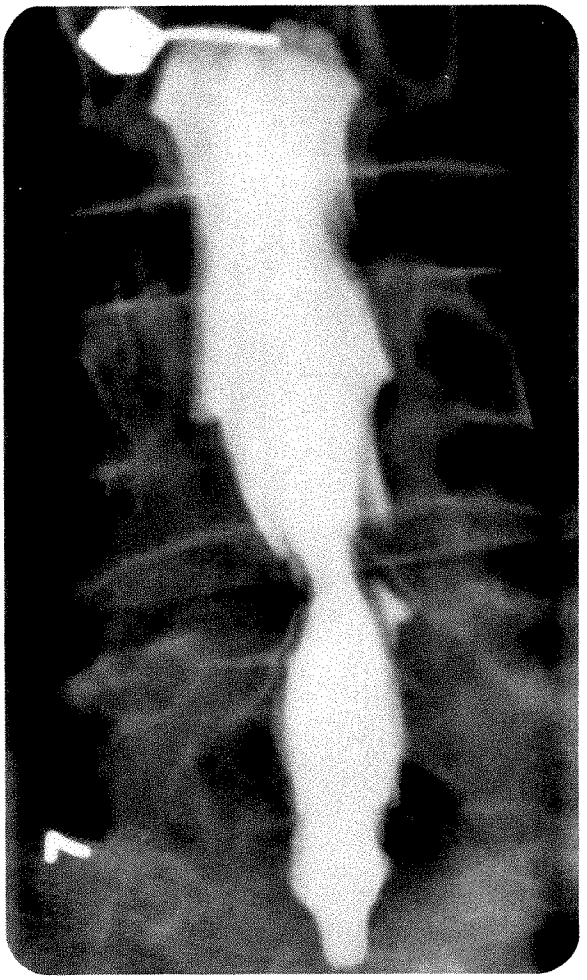
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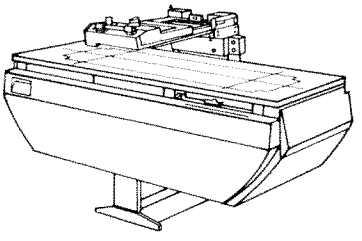
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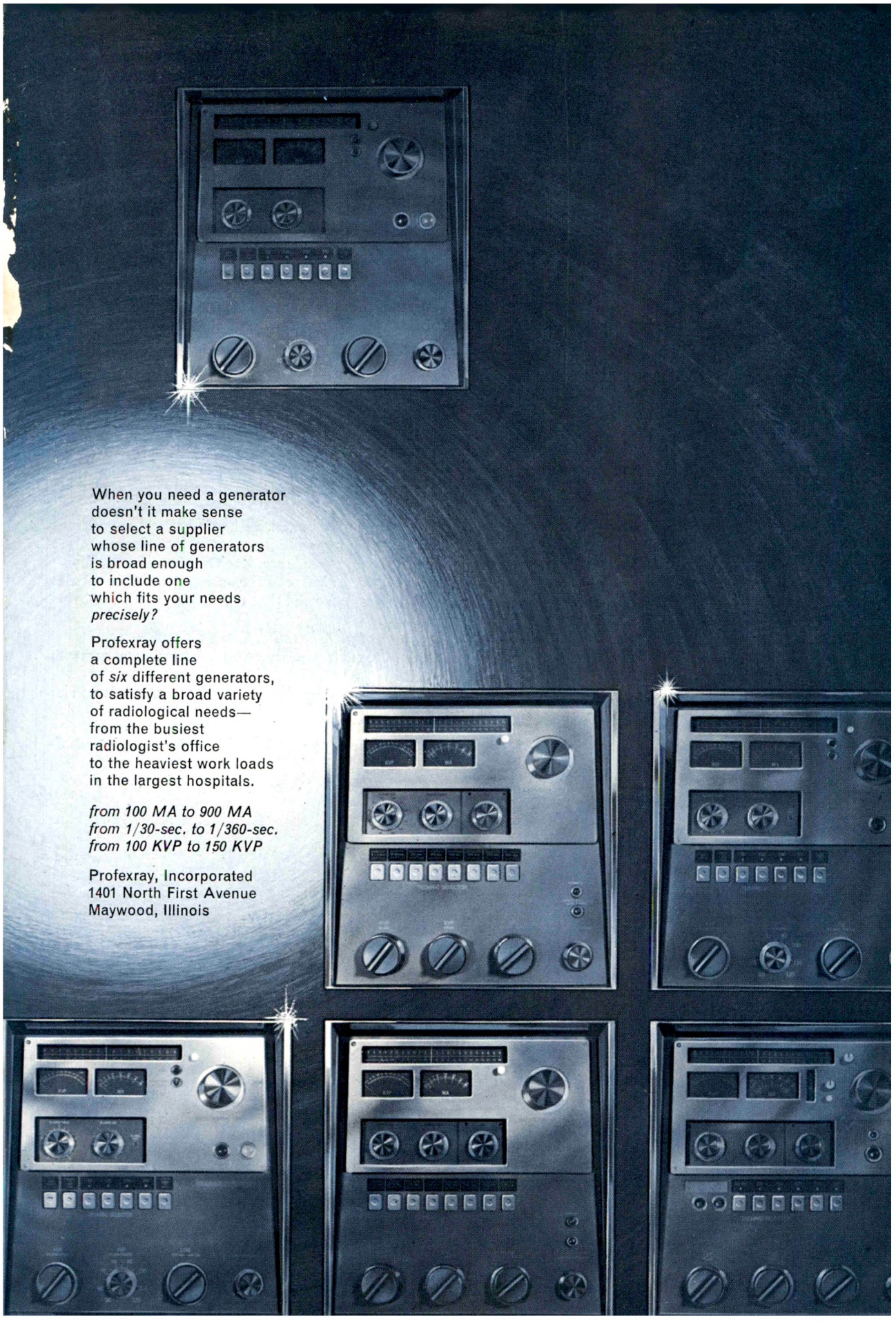


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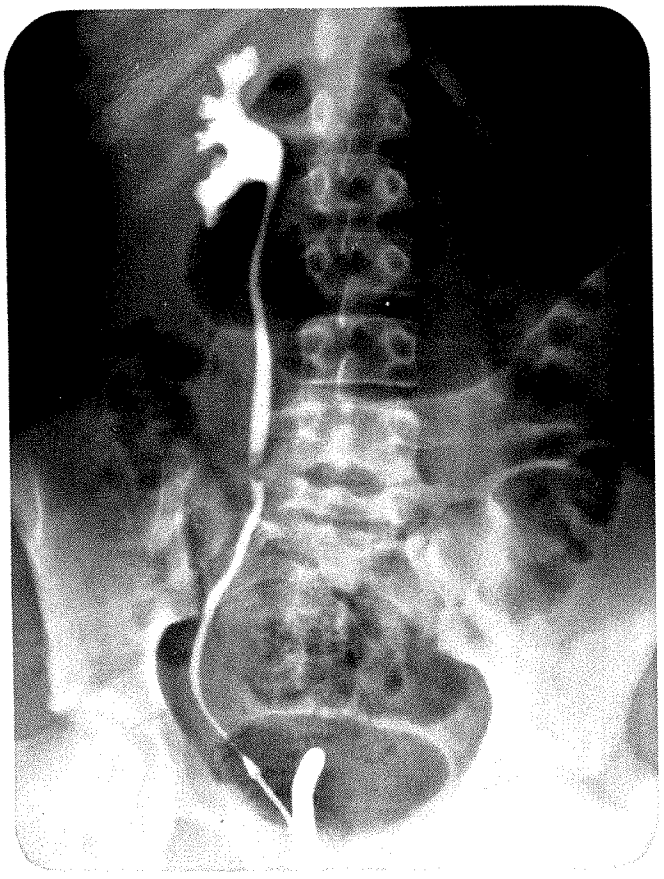
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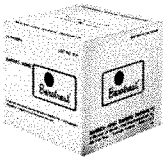
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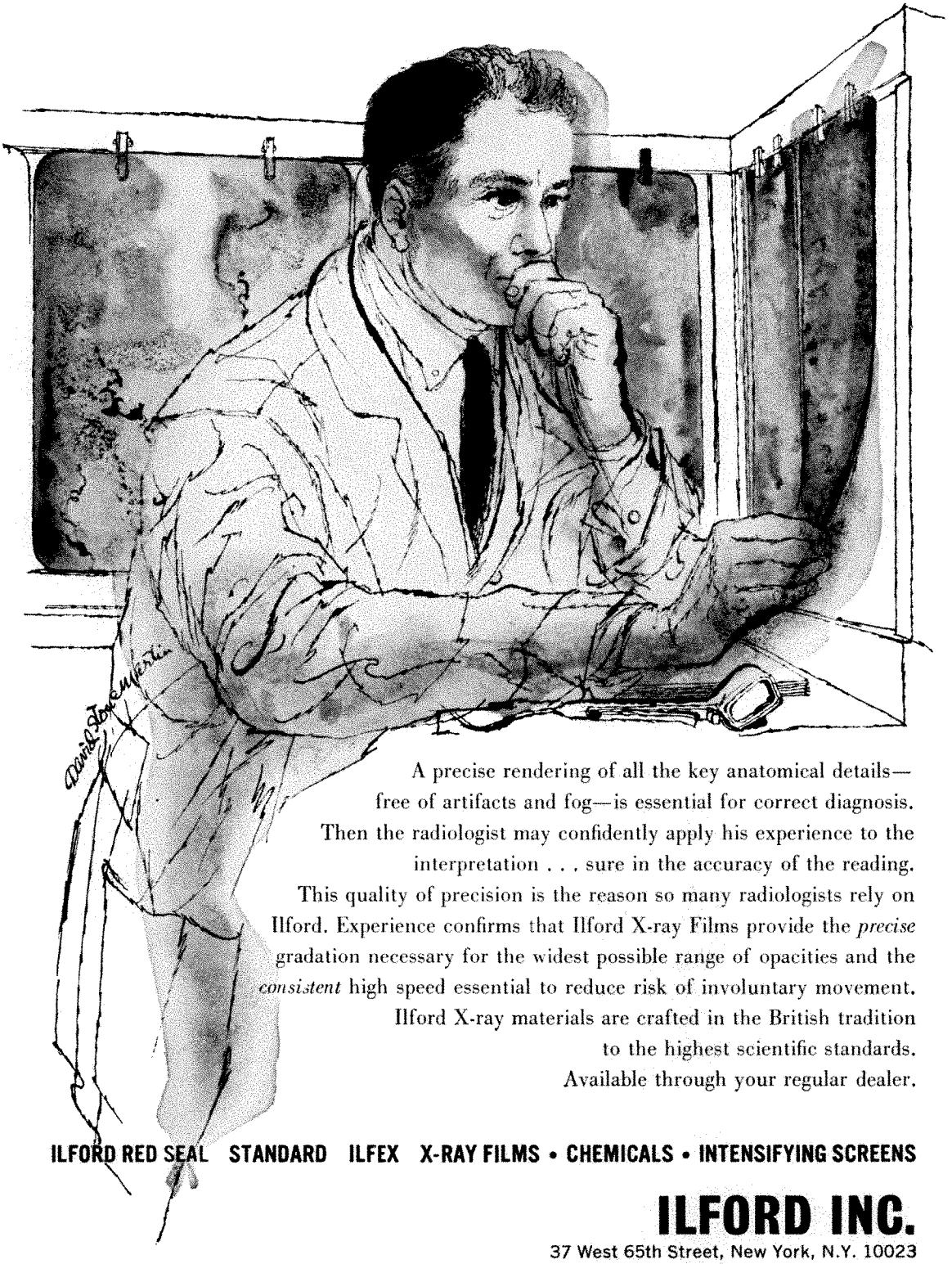
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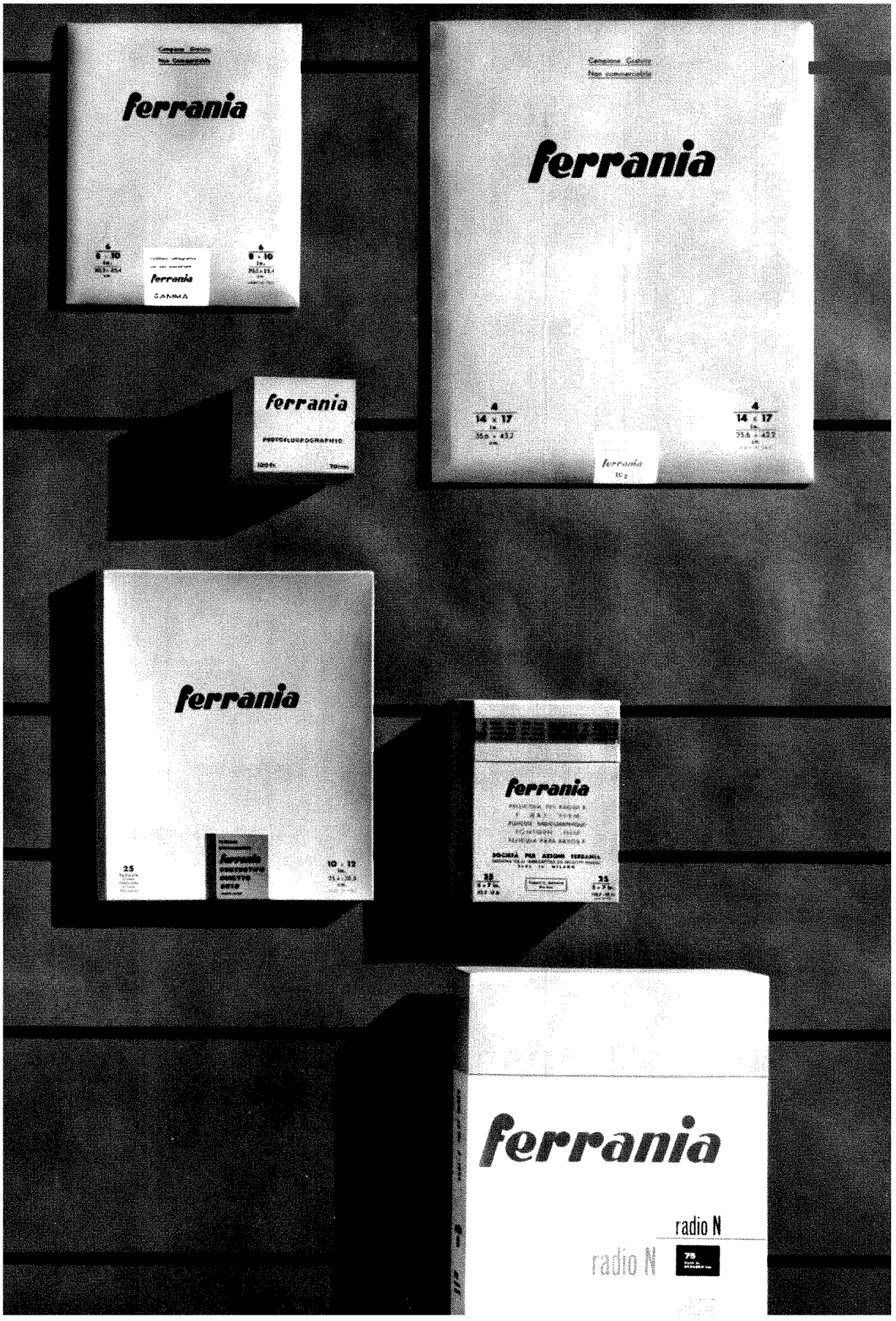
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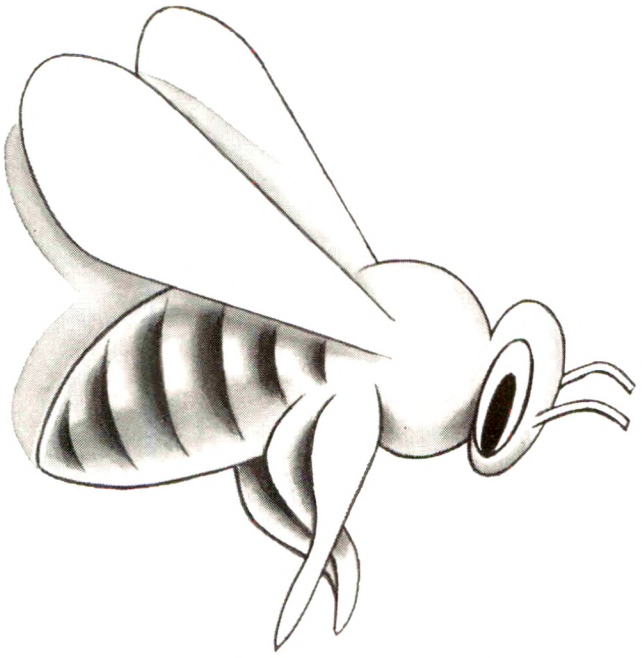
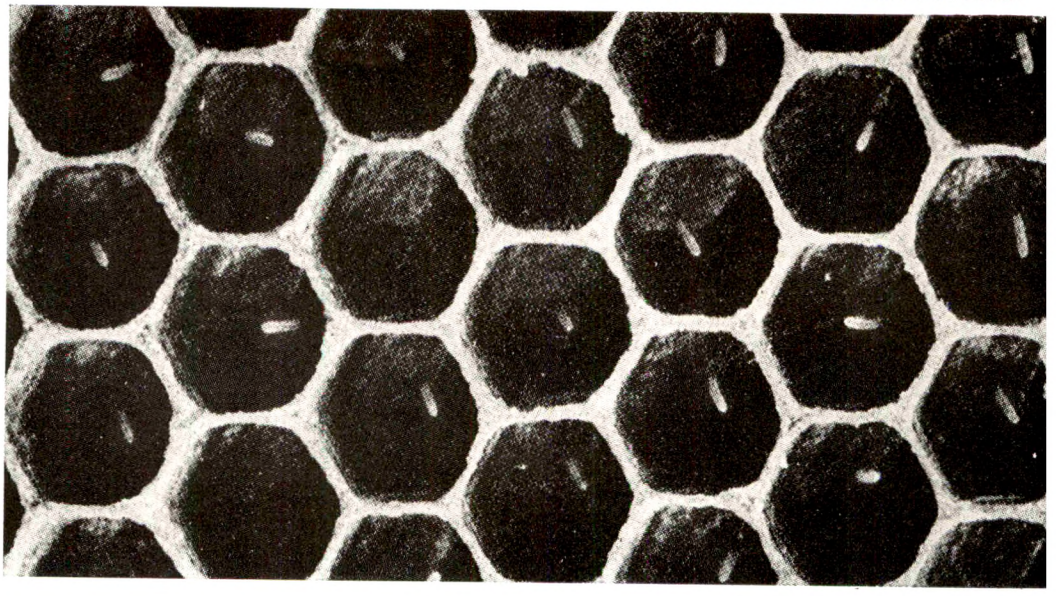


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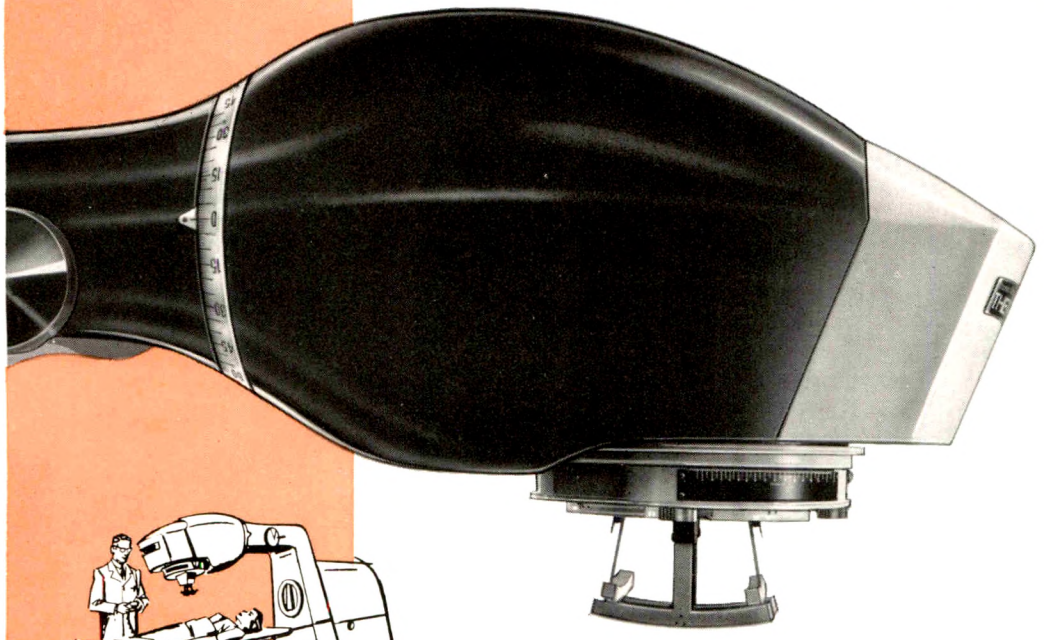
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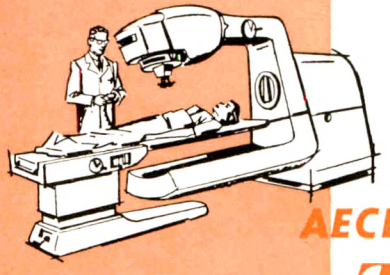
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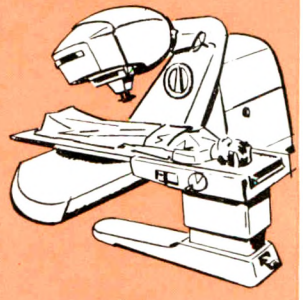




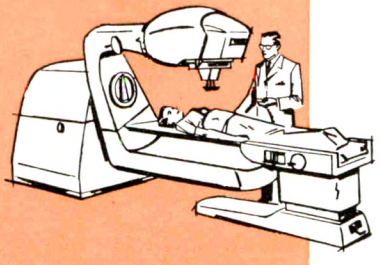
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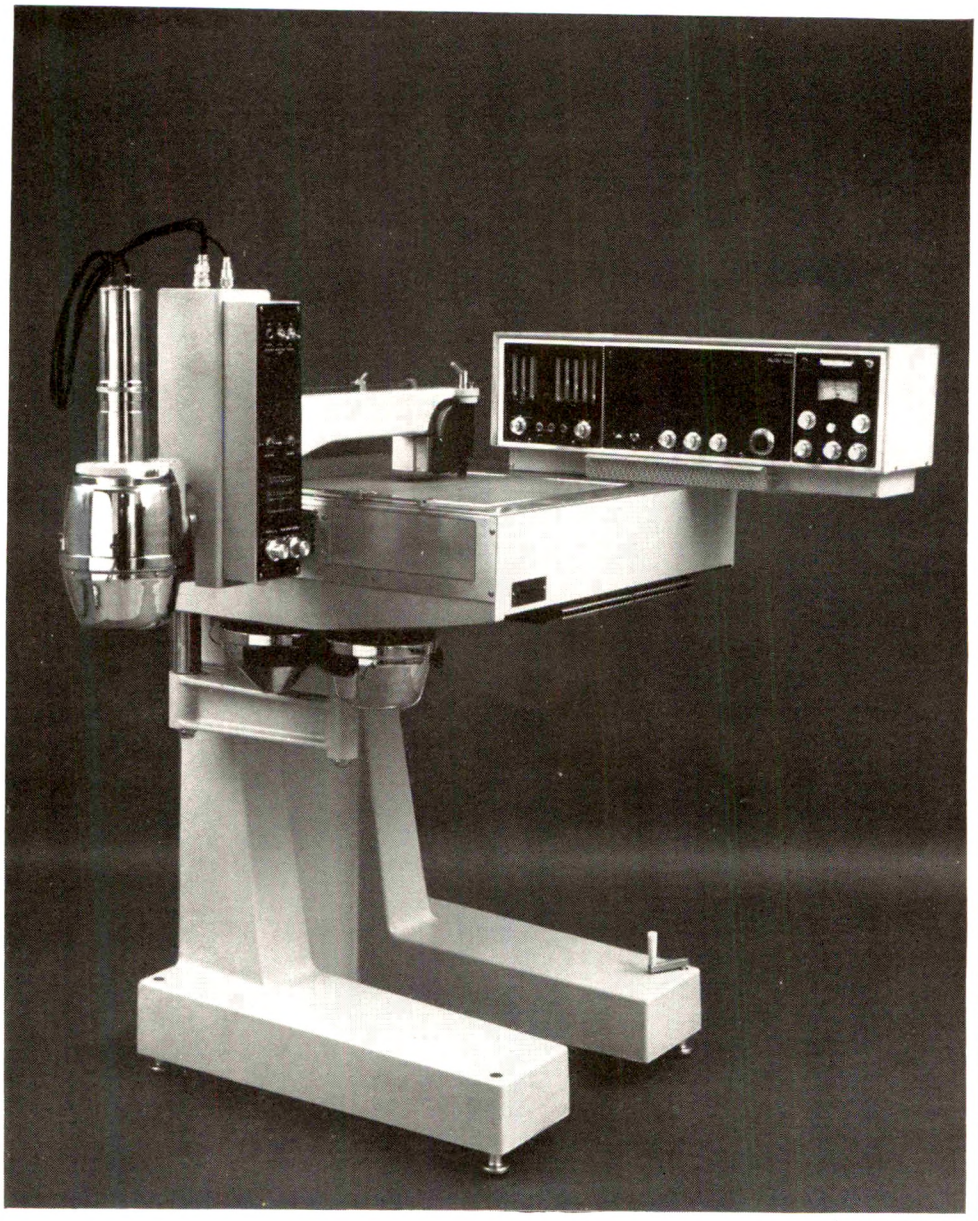
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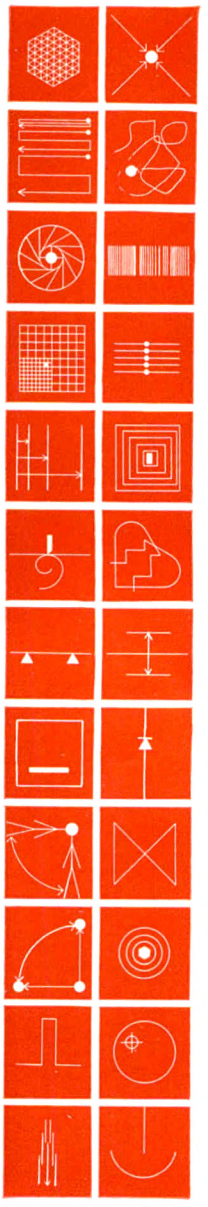
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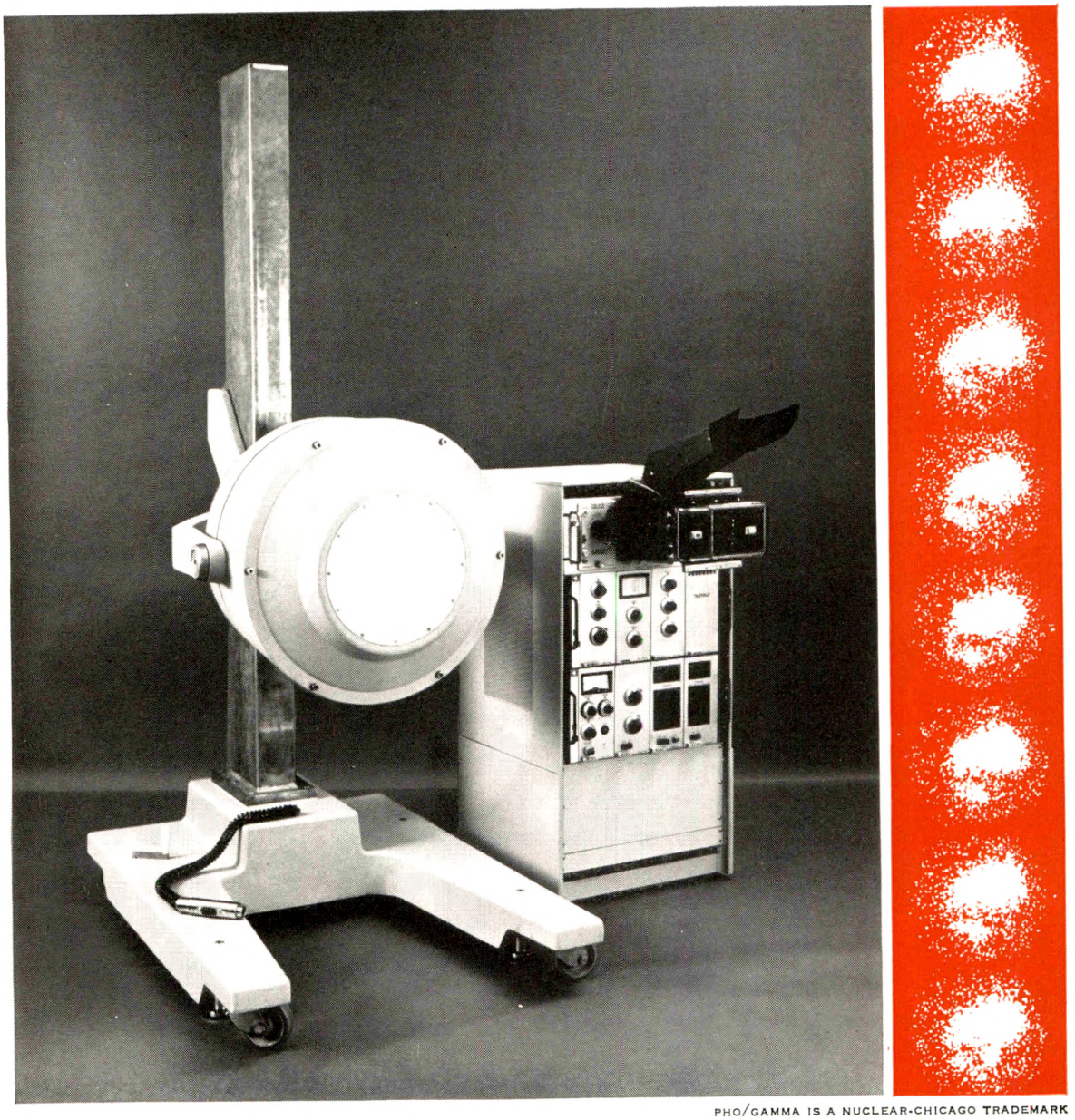
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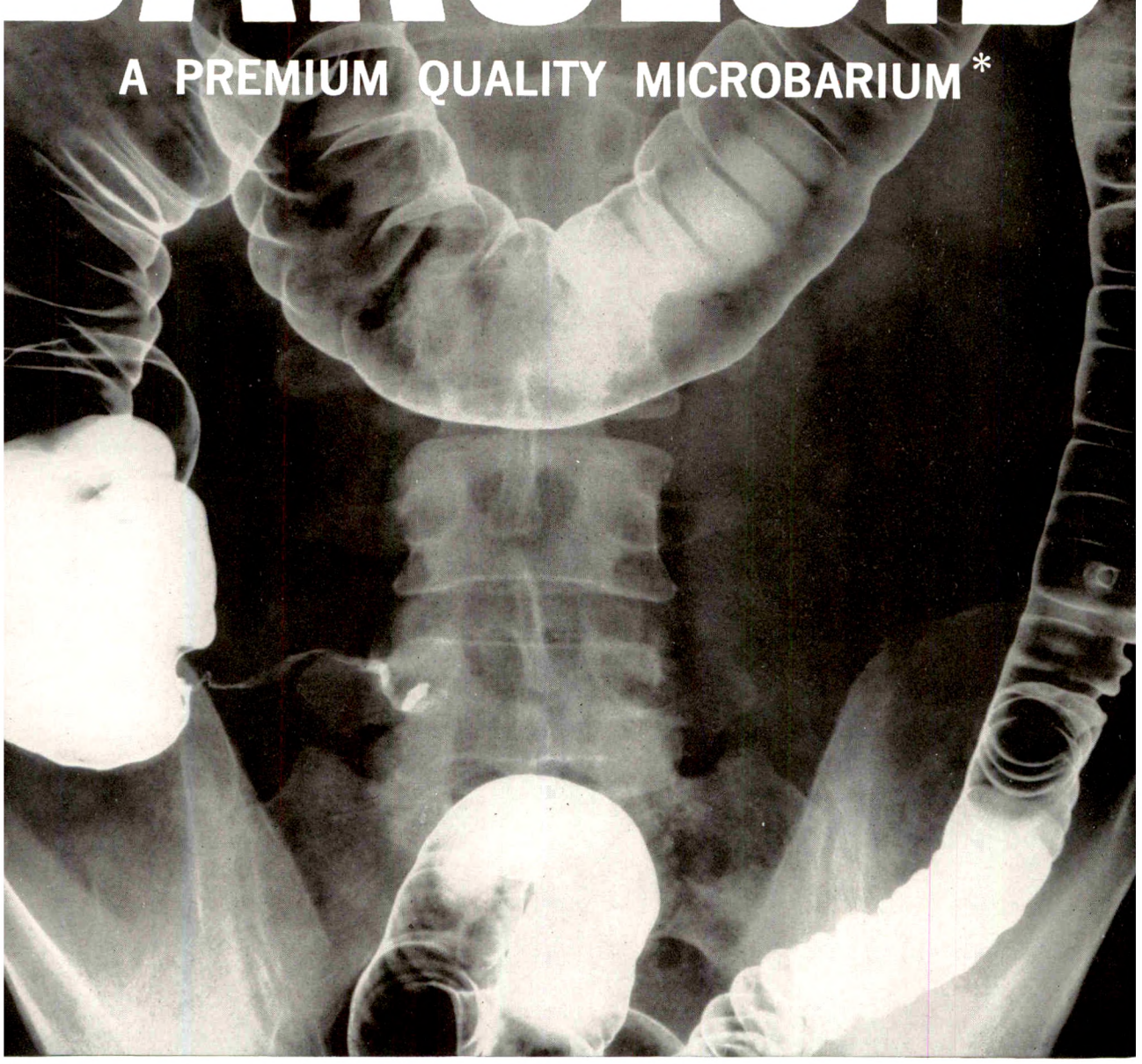
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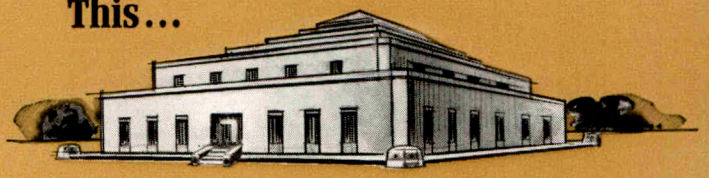
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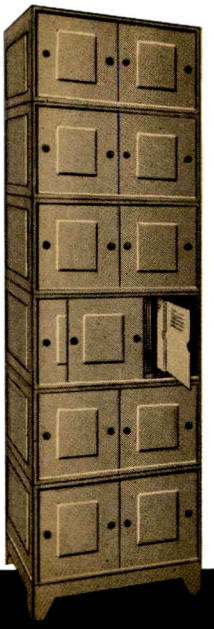
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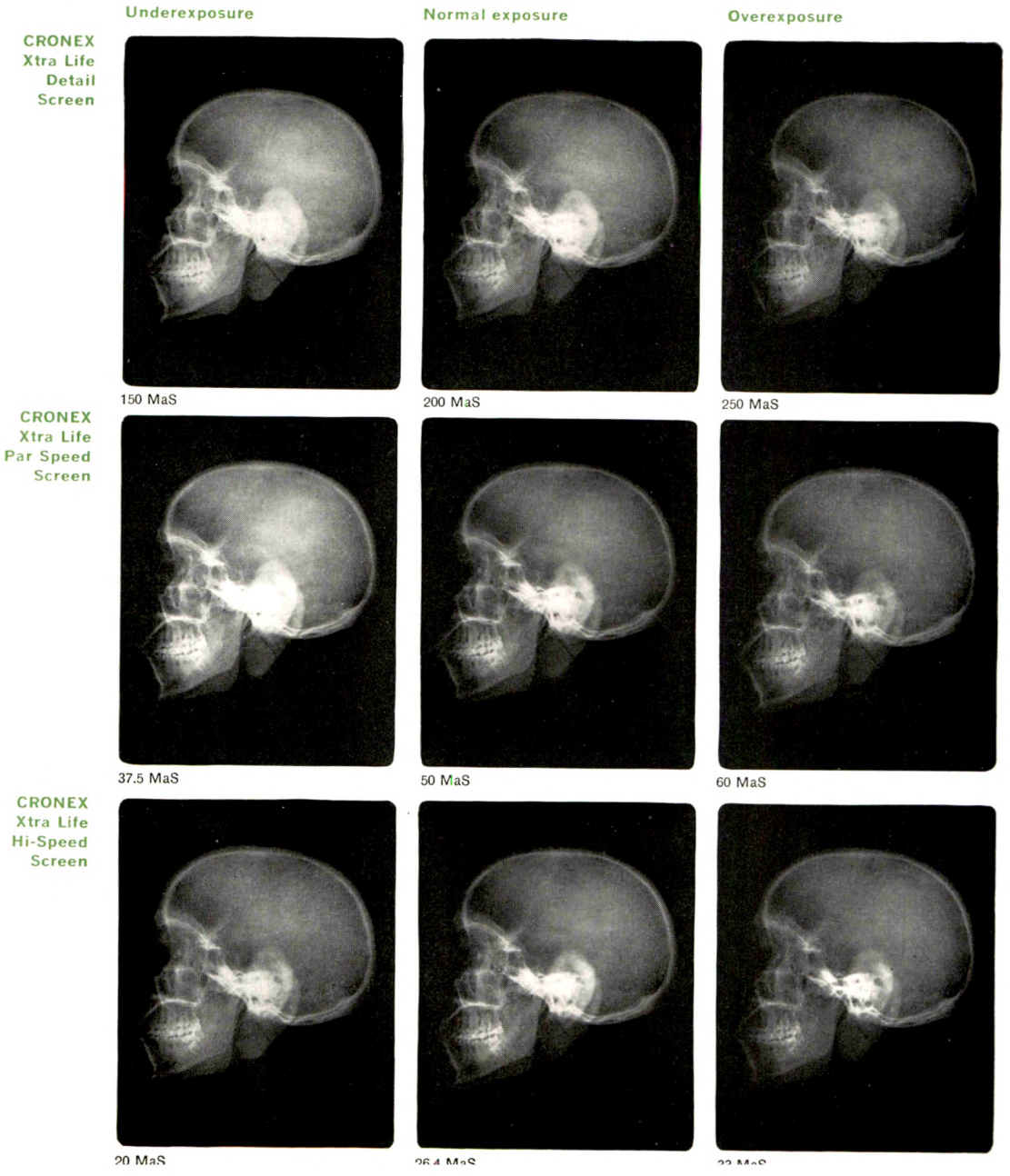
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120 KVP

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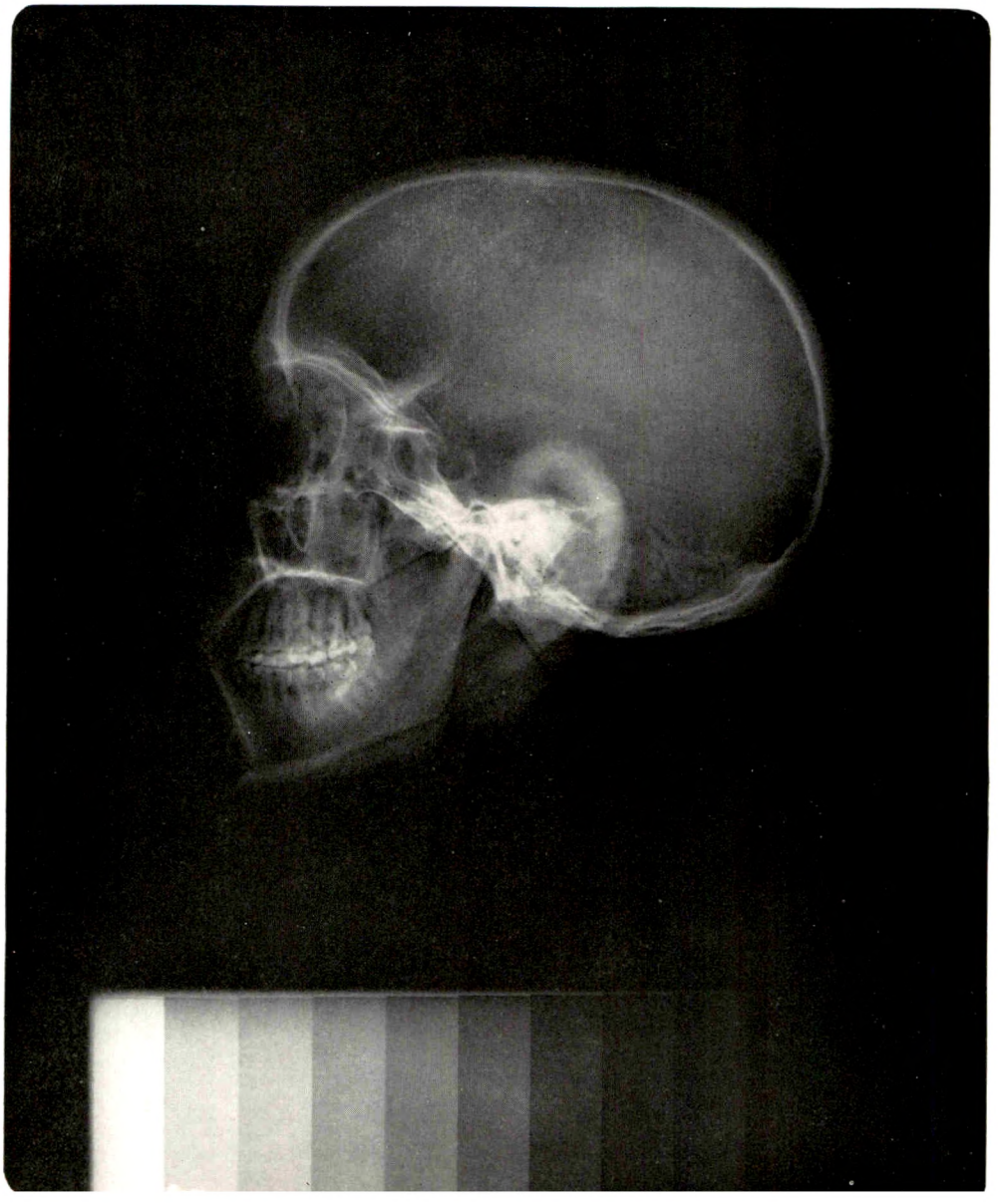
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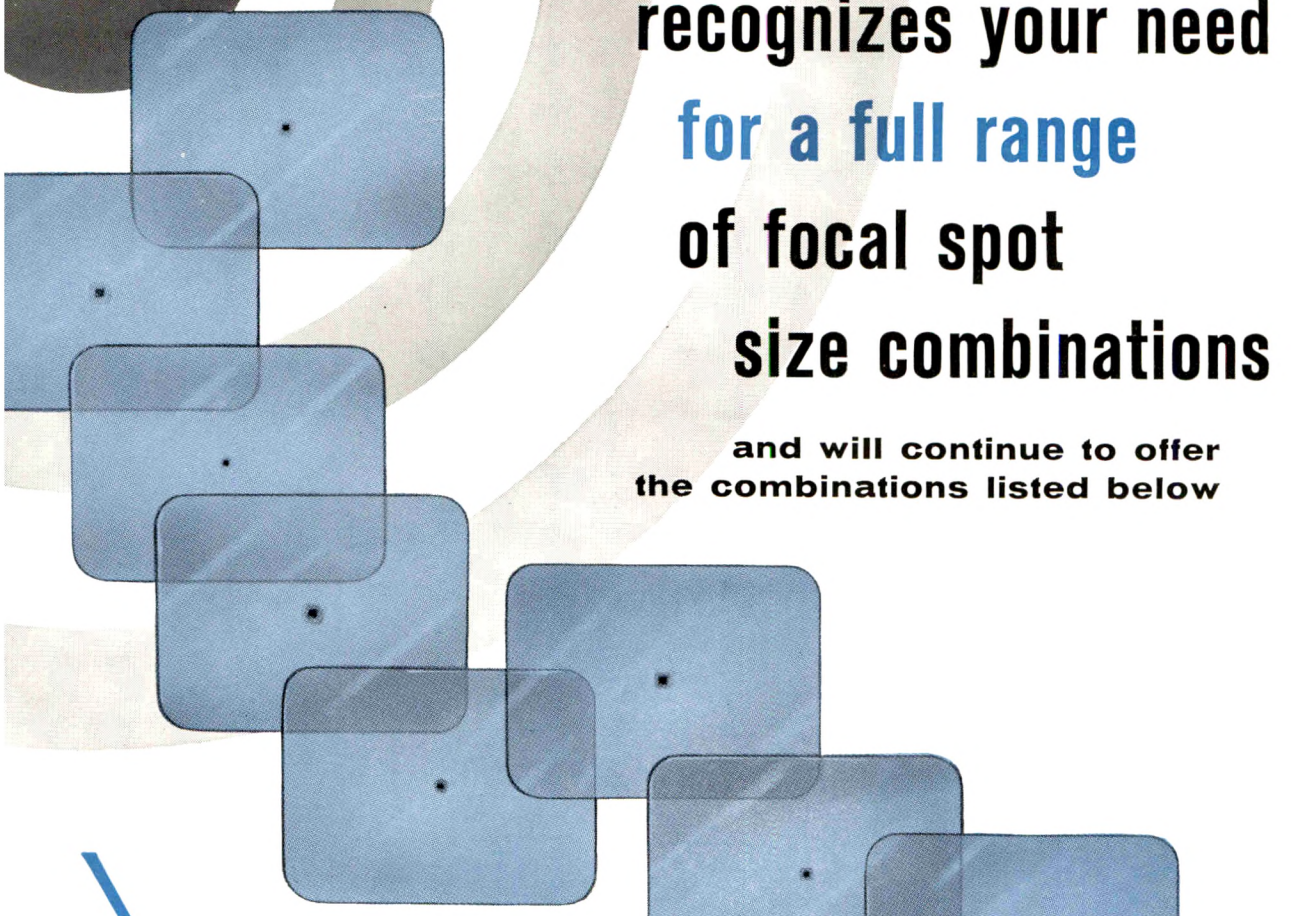
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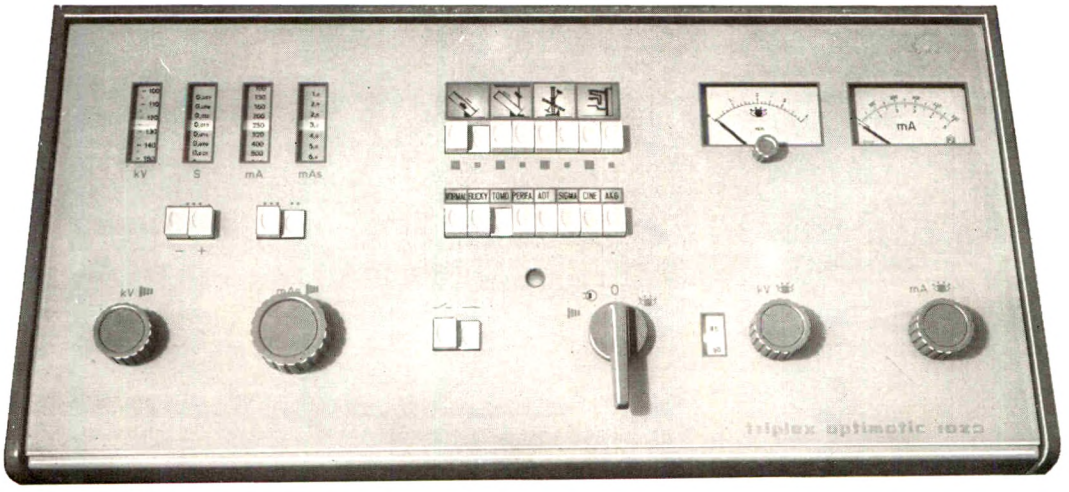
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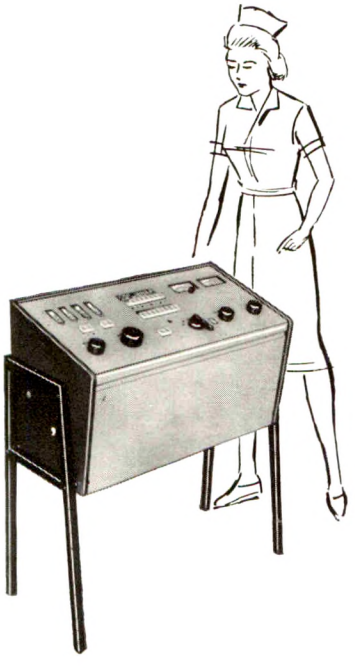
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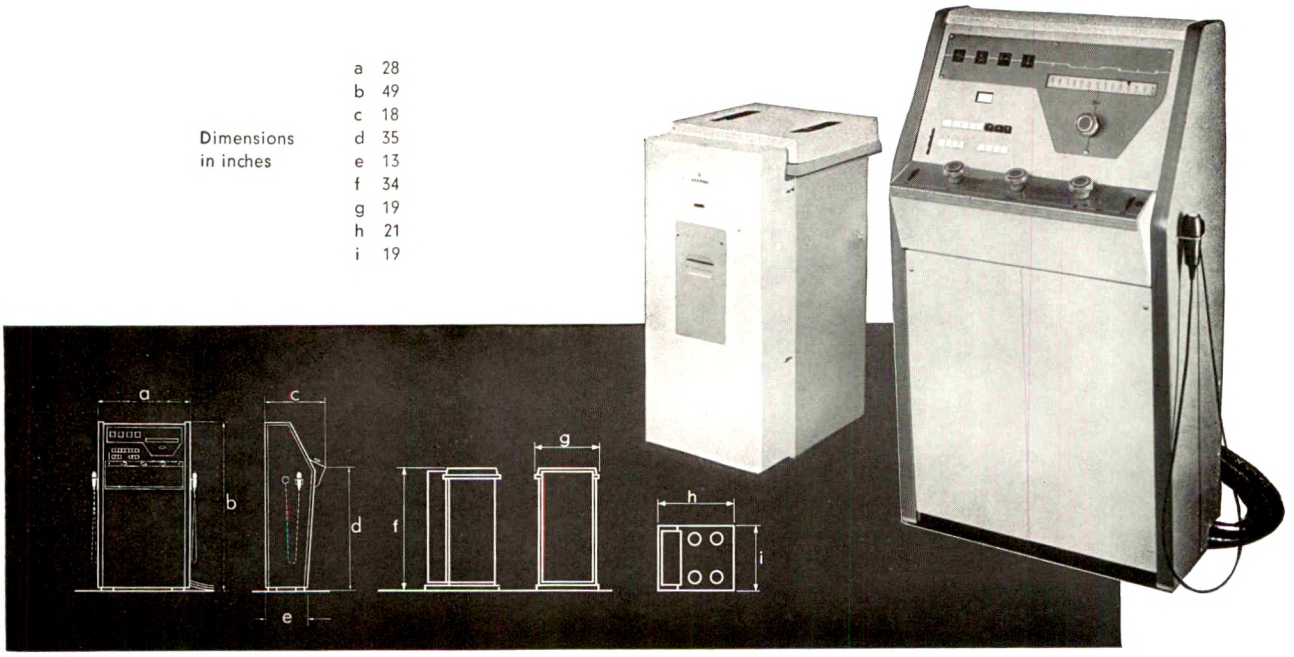
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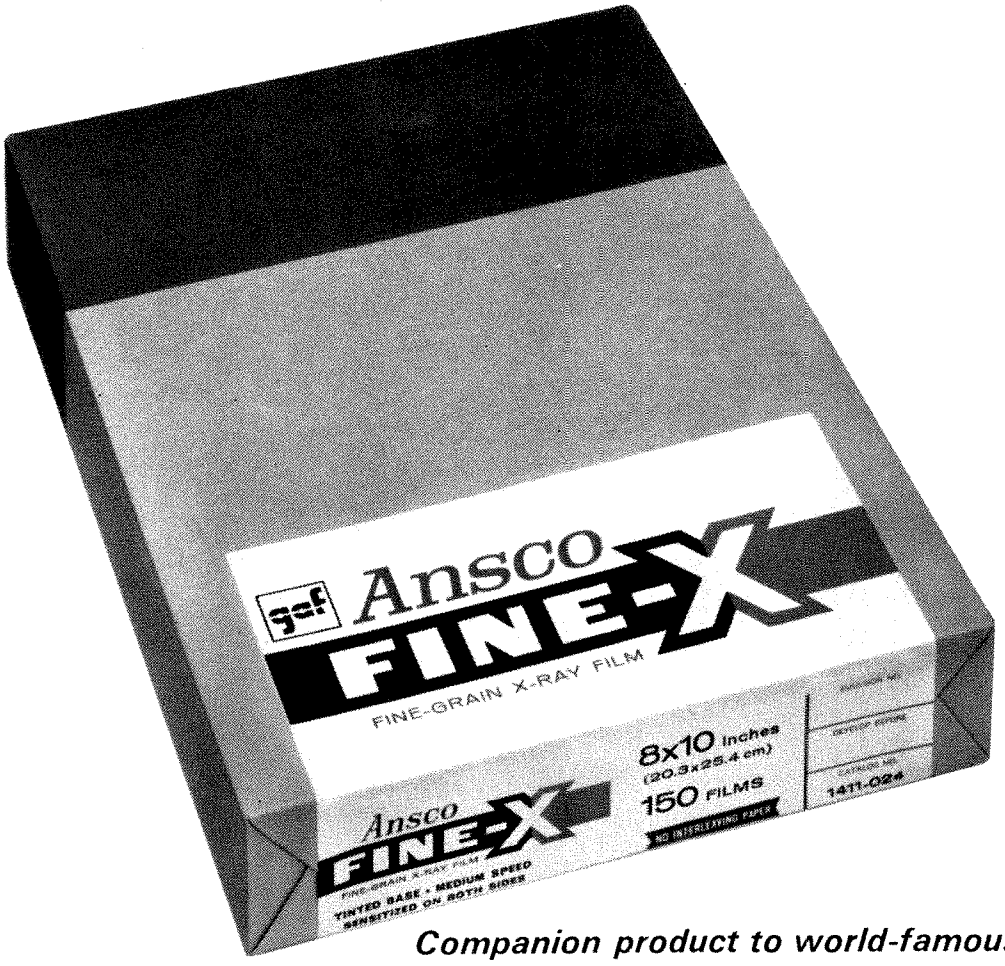
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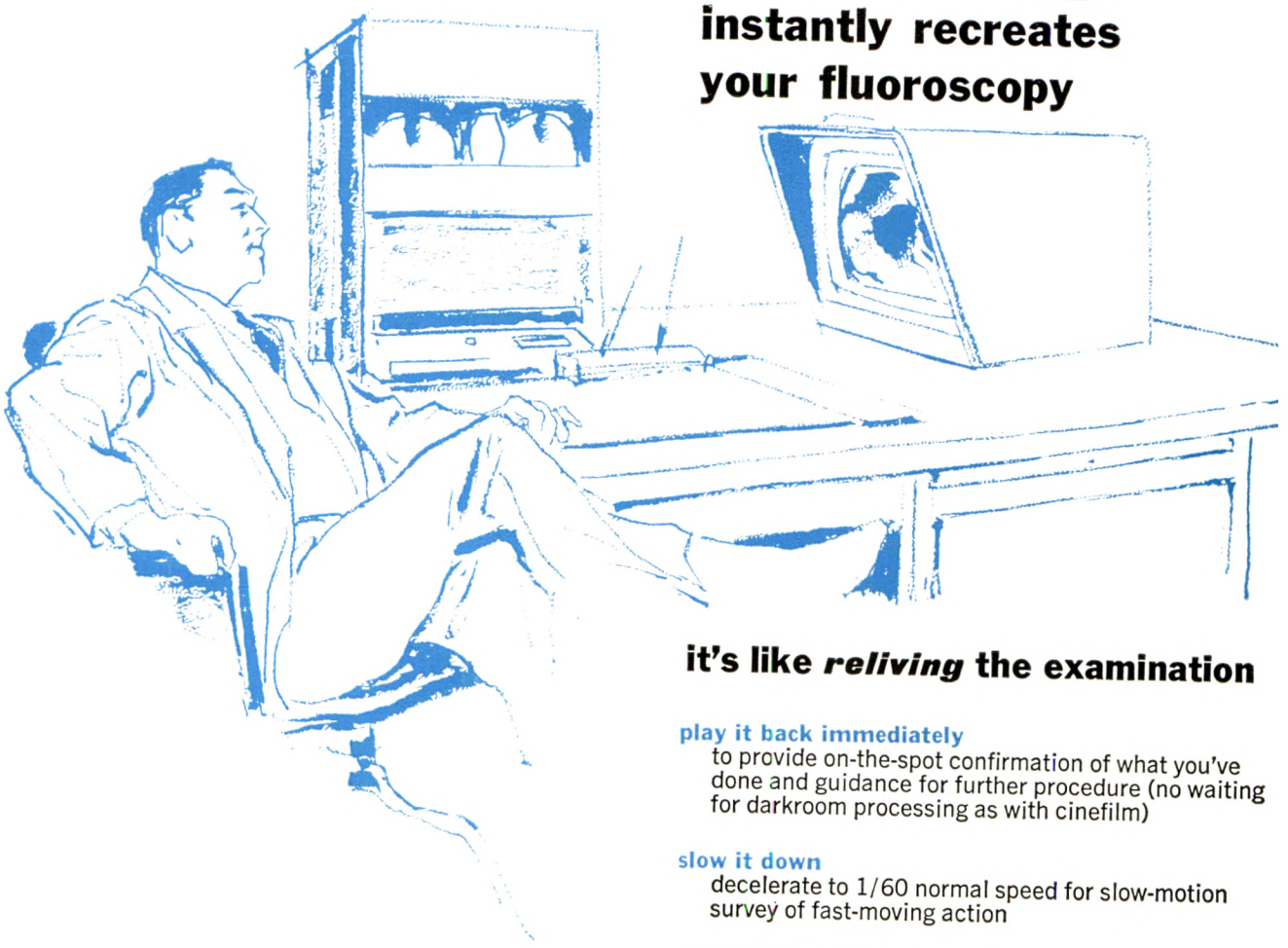
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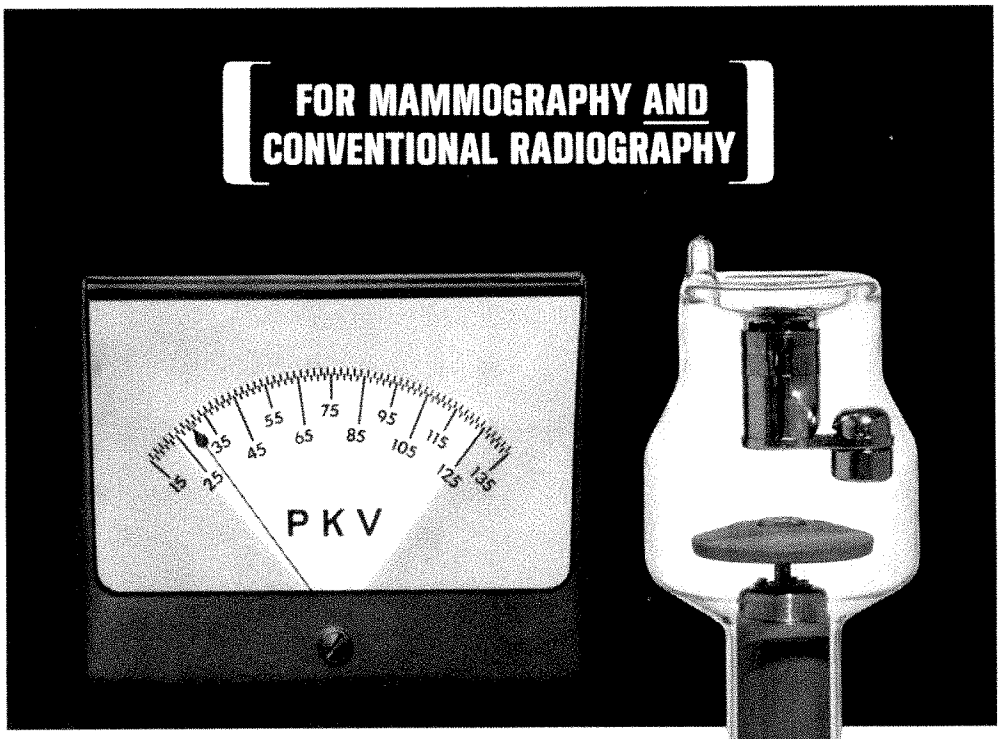
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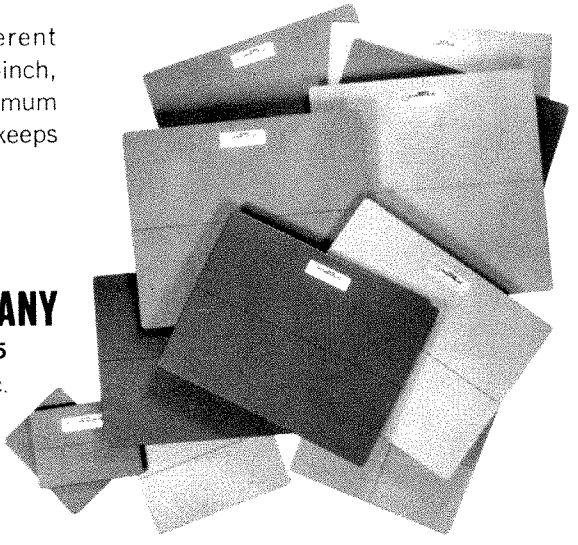
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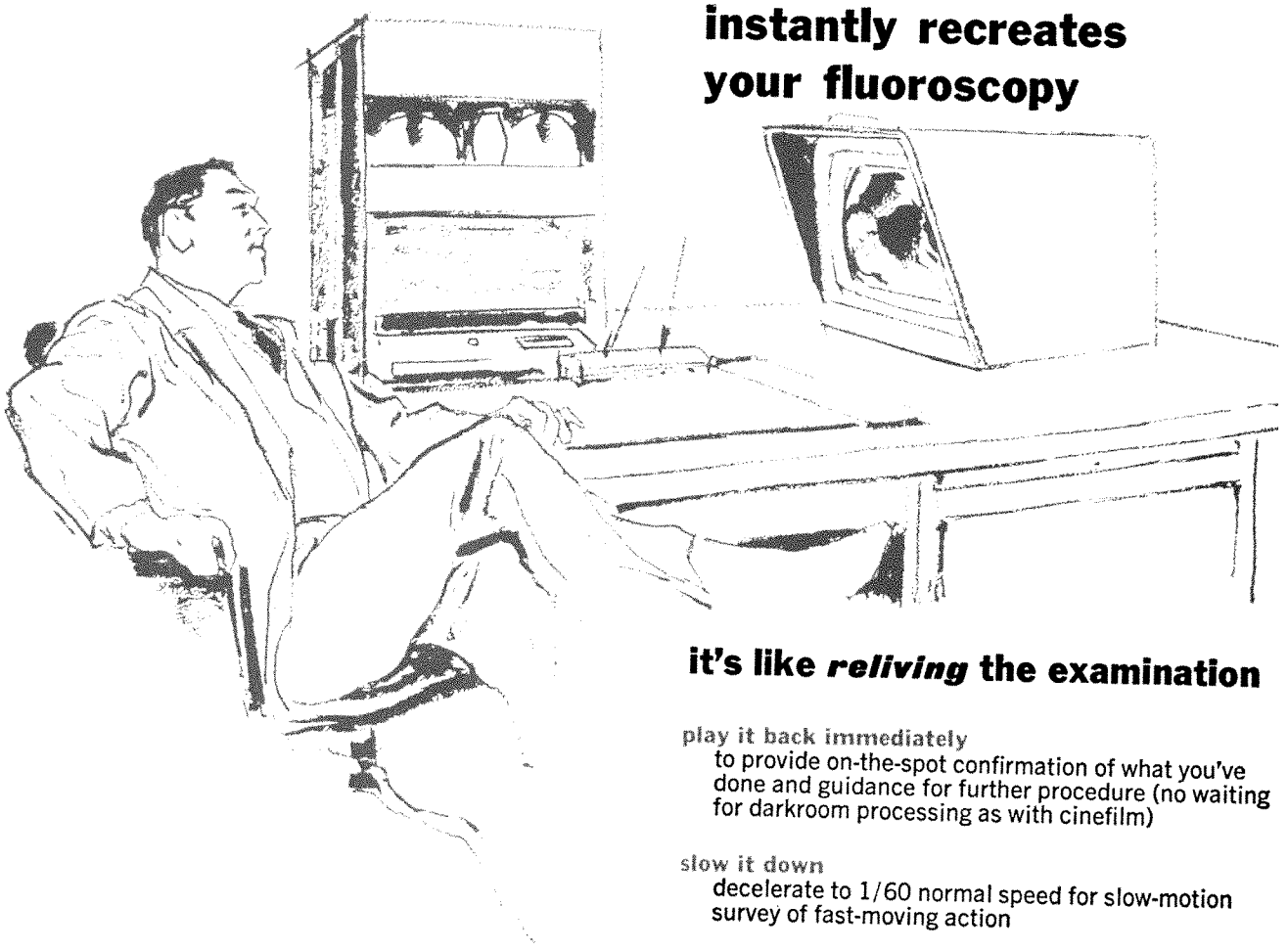
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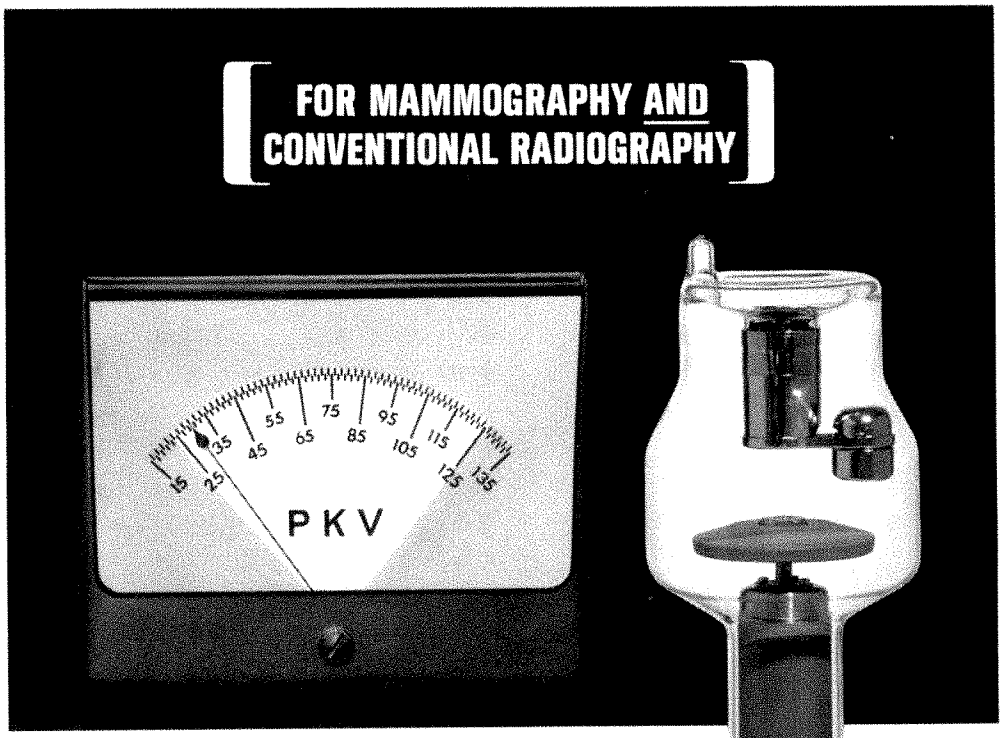
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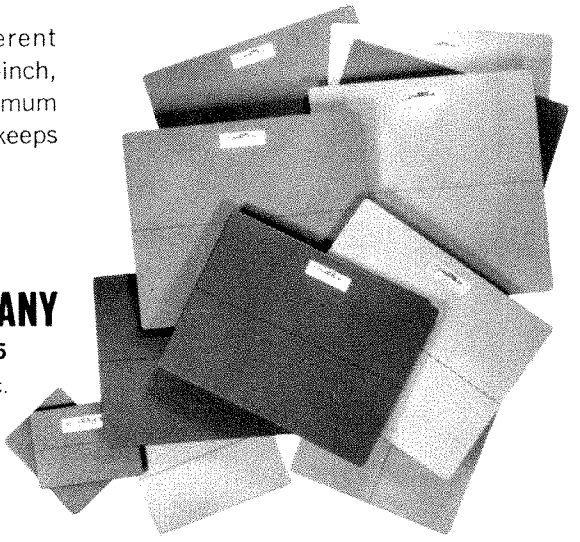
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FREE . . . write for your copy of revised booklet "Characteristics and Applications of X-ray Grids."



L-F

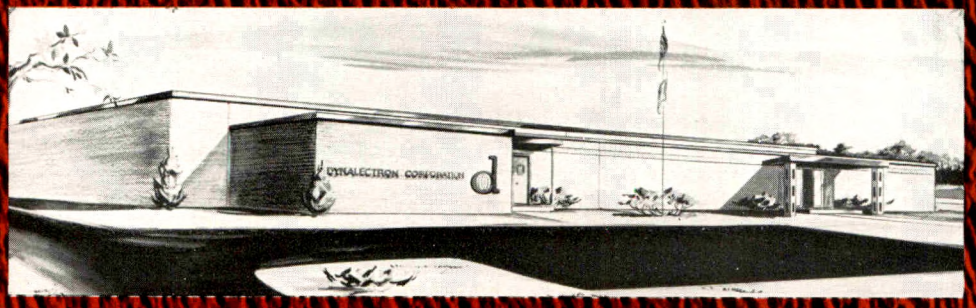
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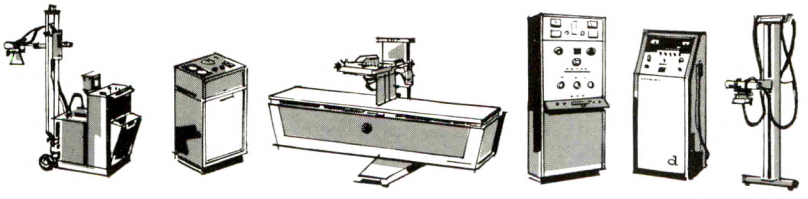
SEE YOUR X-RAY SUPPLIER FOR ALL L-F PRODUCTS

"The difference between failure and success is doing a thing nearly right and doing it exactly right."



We have built a new $1,000,000 plant in which to produce an expanded line of Mattern x-ray apparatus . . . *exactly right!*

Mattern has been a leader in the field of quality x-ray apparatus for more than a third of a century. It's time now that you ask for detailed information about Mattern x-ray tables, control consoles and tubestands, and to see for yourself the tremendous difference between nearly right and exactly right.



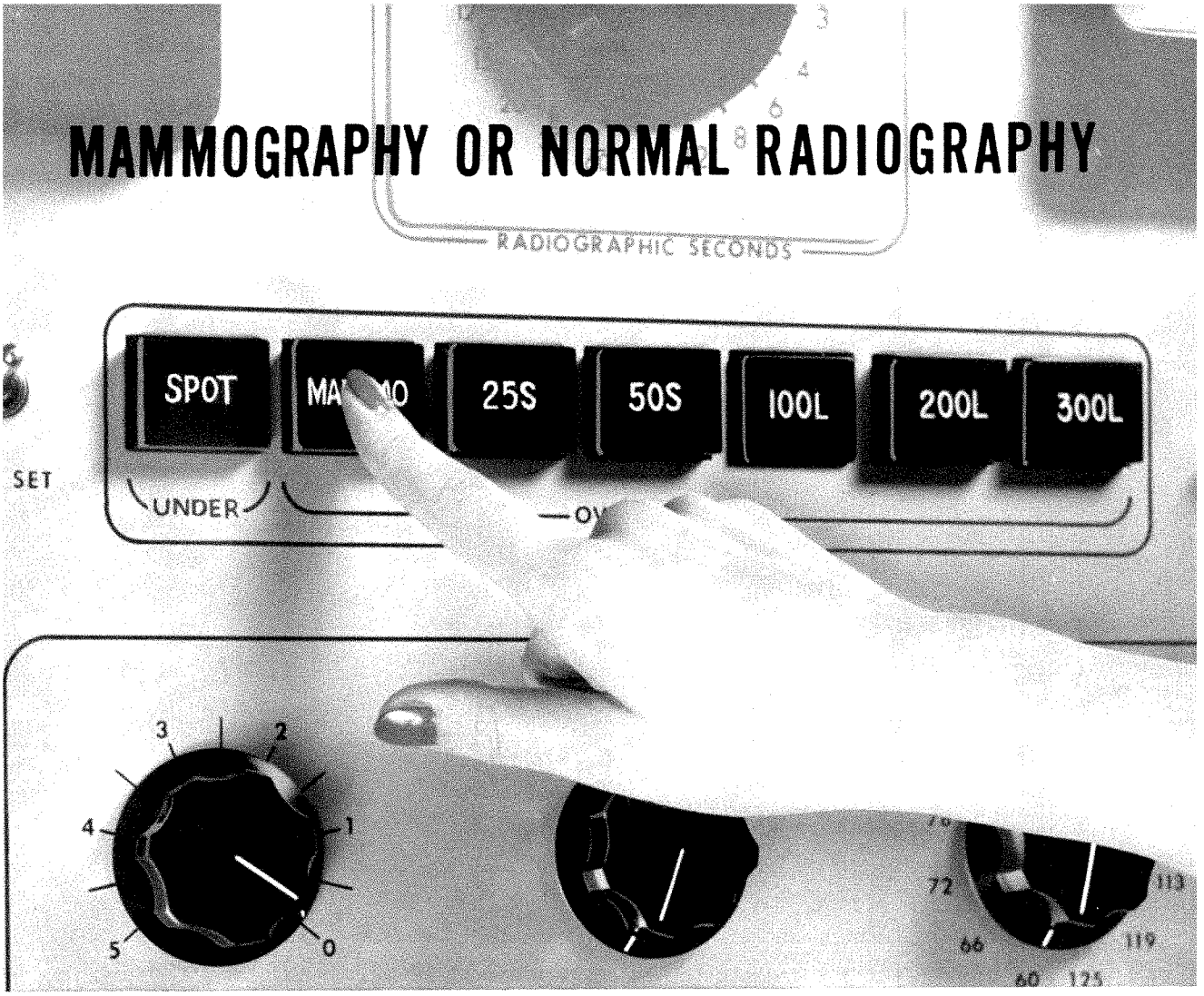
WRITE TO US FOR DETAILED INFORMATION ABOUT X-RAY TABLES, CONTROL CONSOLES AND TUBESTANDS—AUTHORIZED DEALERS THROUGHOUT THE COUNTRY

MATTERN X-RAY

Lake City, South Carolina

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MAMMOGRAPHY OR NORMAL RADIOGRAPHY



IT'S AS EASY AS THIS

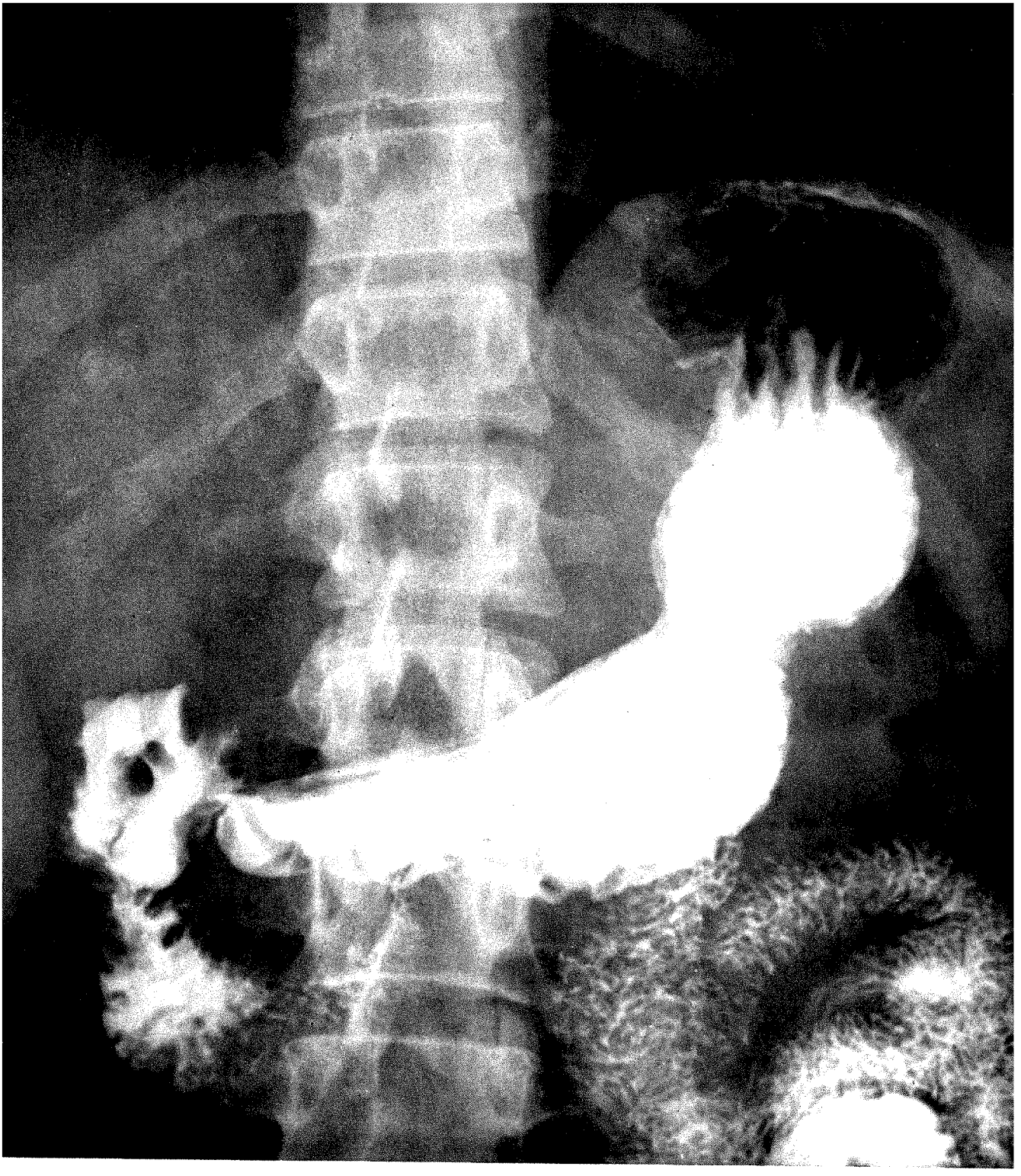
You can do mammography and normal radiography with Continental dual use equipment. These mammography units give you a conventional 300 MA X-ray unit *plus* safe mammography using the following technique factors for mammographic work:

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- **28-30 KVP**
- **0.5 mm focal spot**
- **¼ mm aluminum filter**
- **Beryllium window rotating anode tube**

Efficient X-ray production in both the high and low KV ranges is obtained by using our patented double bridge rectifier circuit. A push button switch changes the transformer power from a series connected, 100 KVP maximum output to a parallel connected, 50 KVP maximum output for efficient 25 to 35 KVP power. Continental dual-use mammography units are available as: Mobile equipment, General Diagnostic equipment or Verta-Vue apparatus.

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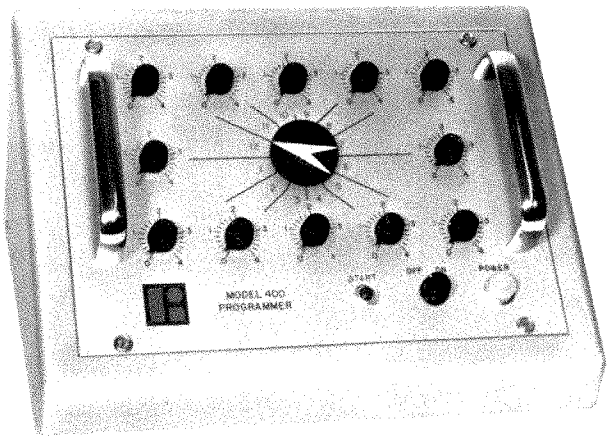
■ Passes from the stomach to the duodenum in a smooth flow through the pyloric sphincter. The duodenal cap is completely delineated to illustrate possible craters or ulcerations very clearly under fluoroscopy.

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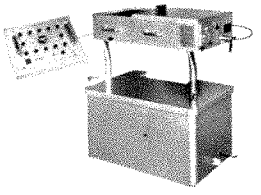
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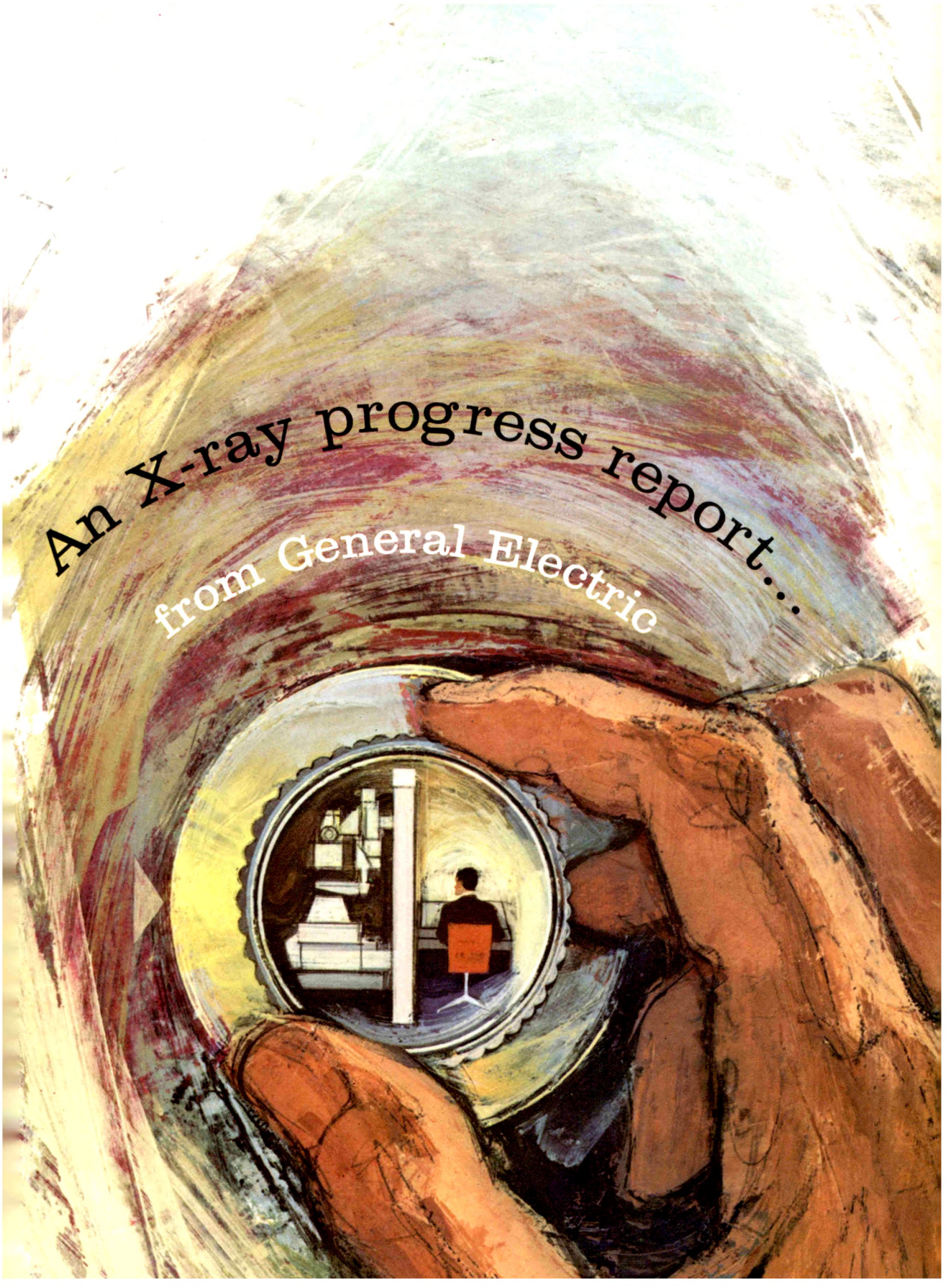
NEW MODEL 400 PROGRAMMER

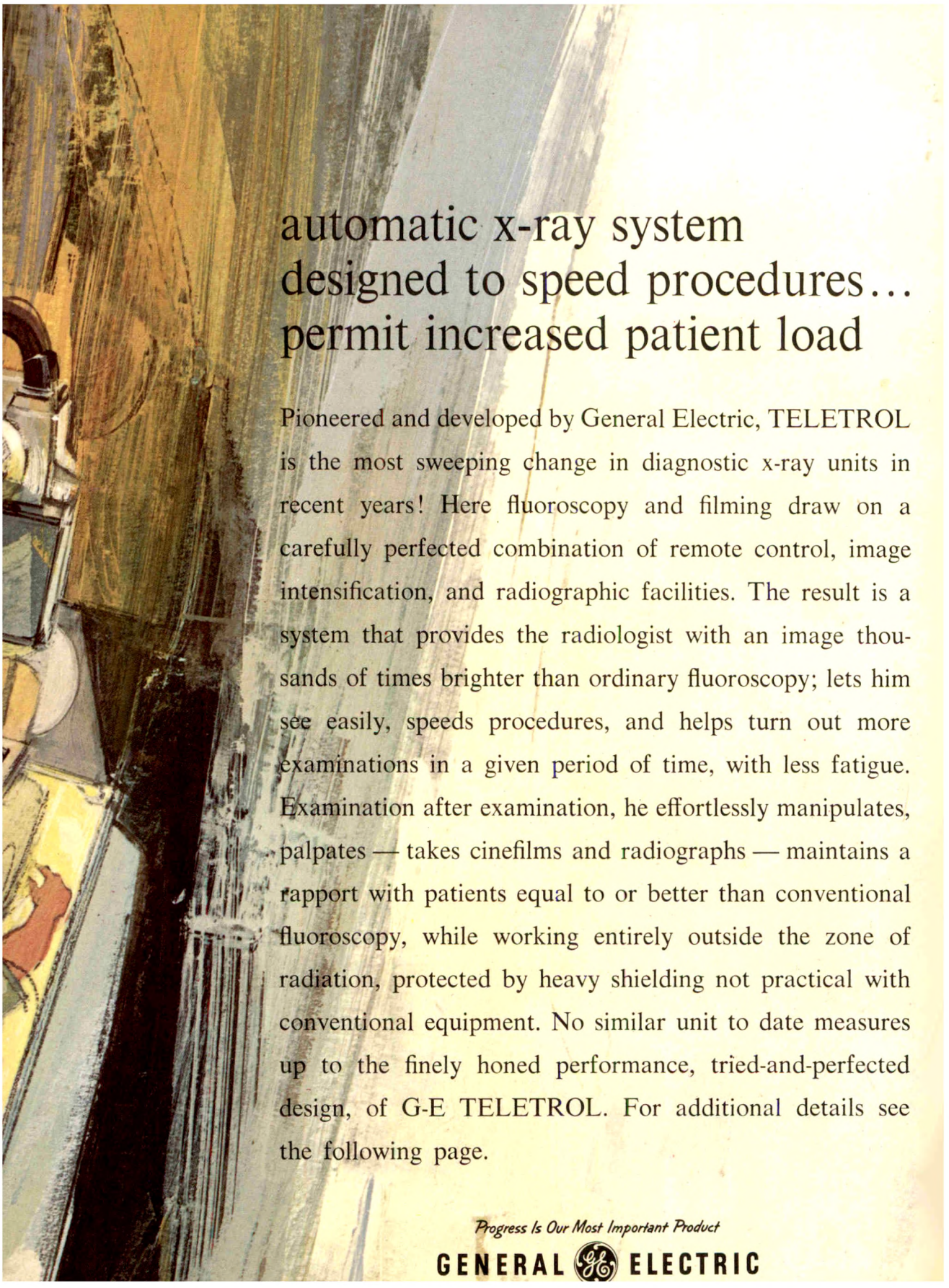
for the Sanchez-Perez Universal Automatic Seriograph. Convenience, an outstanding feature of the Universal Automatic Seriograph, acquires even greater significance with the addition of the Model 400 Programmer. Designed to be plugged into the Seriograph without modification, the Model 400 provides: • Complete series time selection for the high-speed, 12-cassette Automatic Seriograph • Individual time-interval control of each cassette • Preselection — and reselection — of time intervals • Control from remote site • Integral cassette counter • Portability for desk or wall operation • Easy adaptability to automatic dye injectors • Simple adaptability to biplane operation. • Obtain complete information about the convenience-enhancing Model 400 Programmer.



Please address your inquiries to: The Automatic Seriograph Corp., 4900 Calvert Rd., College Park, Maryland. Our telephone number is: UNion 4-5678.

THE AUTOMATIC SERIOGRAPH CORPORATION
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An X-ray progress report...
from General Electric

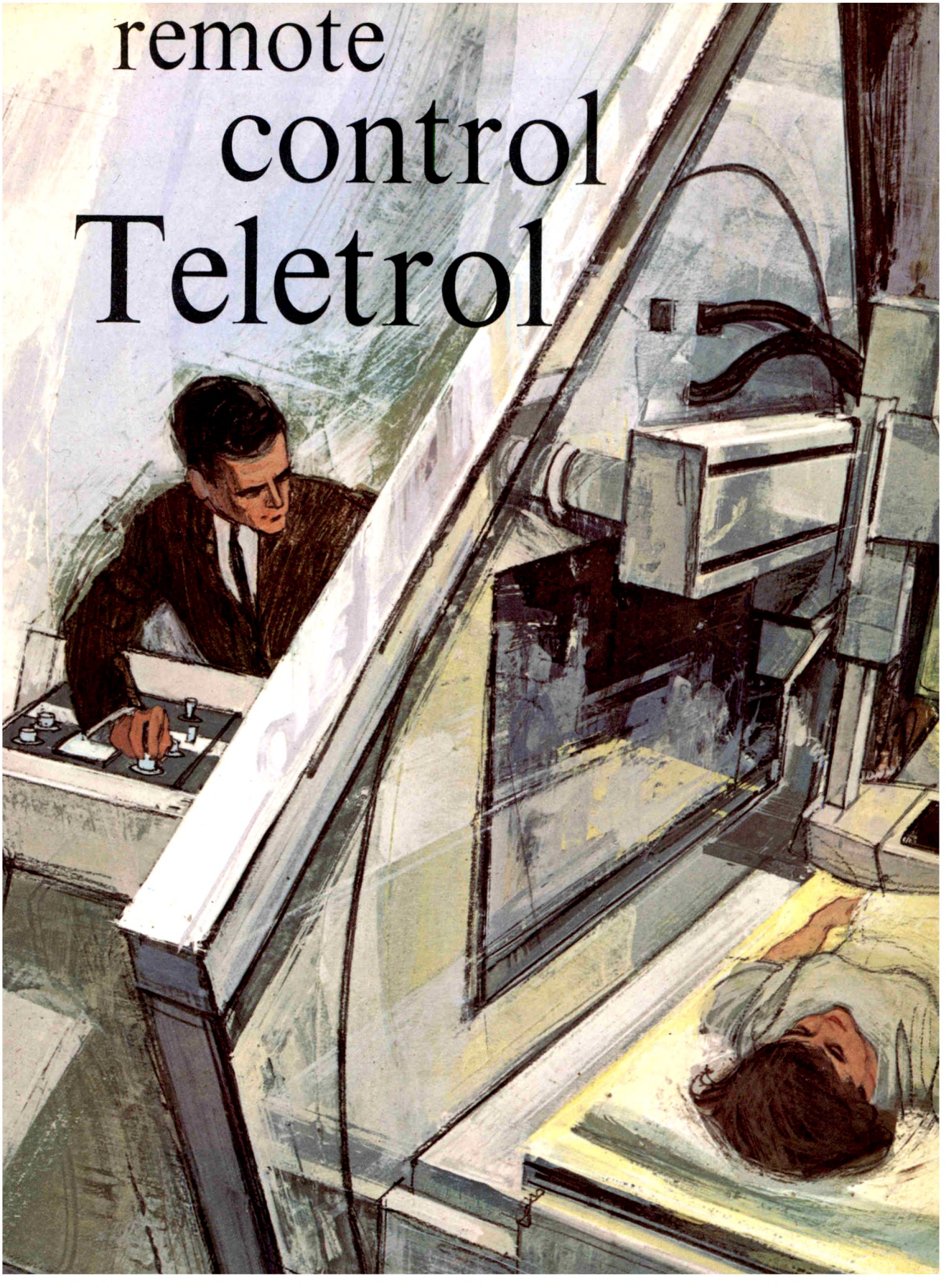


automatic x-ray system designed to speed procedures... permit increased patient load

Pioneered and developed by General Electric, TELETROL is the most sweeping change in diagnostic x-ray units in recent years! Here fluoroscopy and filming draw on a carefully perfected combination of remote control, image intensification, and radiographic facilities. The result is a system that provides the radiologist with an image thousands of times brighter than ordinary fluoroscopy; lets him see easily, speeds procedures, and helps turn out more examinations in a given period of time, with less fatigue. Examination after examination, he effortlessly manipulates, palpates — takes cinefilms and radiographs — maintains a rapport with patients equal to or better than conventional fluoroscopy, while working entirely outside the zone of radiation, protected by heavy shielding not practical with conventional equipment. No similar unit to date measures up to the finely honed performance, tried-and-perfected design, of G-E TELETROL. For additional details see the following page.

Progress Is Our Most Important Product

GENERAL ELECTRIC


remote
control
Teletrol

PERFORMANCE WRAP-UP...
Facts on TELETROL remote control x-ray

General Electric TELETROL completely integrates all the following automatic features into one unified diagnostic system:

Remote controlled image intensifier — Feeds into control-console TV monitor, providing images easily bright enough for light-room viewing. Employs full 9-inch image intensifier tube. Additional TV monitors can be installed in other rooms, to facilitate teaching and consultations. A second TV circuit also can link the console to the darkroom or other location so reference films can be viewed right during examinations. Audio intercom allows easy conversation with patient.

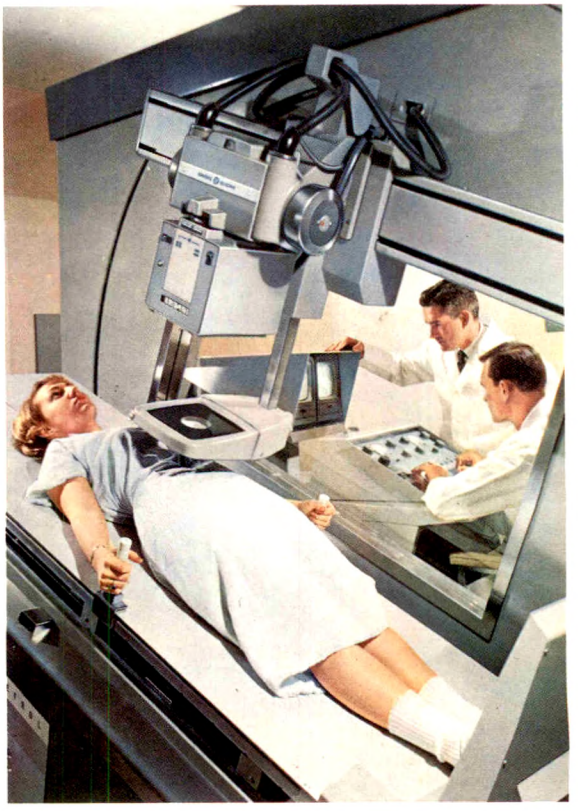
16mm synchronous cinefilm system — Automatic pulse-generator, coupled to shutter, synchronizes exposures to occur only while shutter is open, minimizing radiation to patient.

Remote-controlled table — Angulates 90° either side of horizontal, controlled from console; provides for longitudinal and lateral movement for complete scanning of patient.

Automated x-ray tube unit — Heavy-duty, rotating-anode tube is centered to the intensifier tube and tracks it automatically at all times. Mounted above table it provides 36-inch focal-spot-to-film distance.

Remote-controlled collimator — Shutters fully adjustable, laterally and longitudinally, from single-control on console. Shutter indicators for precise control of field size.

Rotational footrest — Built-in, power-driven turntable allows patients to be turned and positioned by remote control, when in vertical attitude.



Power-operated cassette tray — Accepts any-size standard cassette. Aligns automatically with x-ray tube upon signal from console, while system switches over to preselected radiographic technic.

150-kvp, 300-ma x-ray generator — Operates directly from the TELETROL control console. Includes built-in phototiming for consistent radiographic results.

Total TELETROL performance requires a good deal more description, and must be seen to be fully appreciated. For information, or to arrange a demonstration, see your G-E x-ray representative. Or write to X-ray Department, General Electric Co., Milwaukee 1, Wis.

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- Gives a smooth, even coating to the stomach, small bowel, and colon.
- Will not dry or "flake" in the colon for an hour or more allowing re-examination on same visit.
- Non-foaming, stable formula allows excess suspension to be saved and used another day.
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When the table is vertical, the counterweight is automatically uncoupled so the spot film device can be moved with a finger, literally.

The Norelco Diagnost 50 was designed to make routine examinations easy in every way. It's light, yet strong, with almost no heavy parts of cast metals. Changeover to horizontal and 15 degrees Trendelenburg position is, of course, motor driven, with provision for stopping at any intermediate position.

The narrow table base sweeps back sharply, and no parts project over the base or table edge. Work around the table is easy. Your feet are free of obstruction; you enjoy what we call "foot comfort."

...the Norelco Diagnost 50 gives you fingertip movement



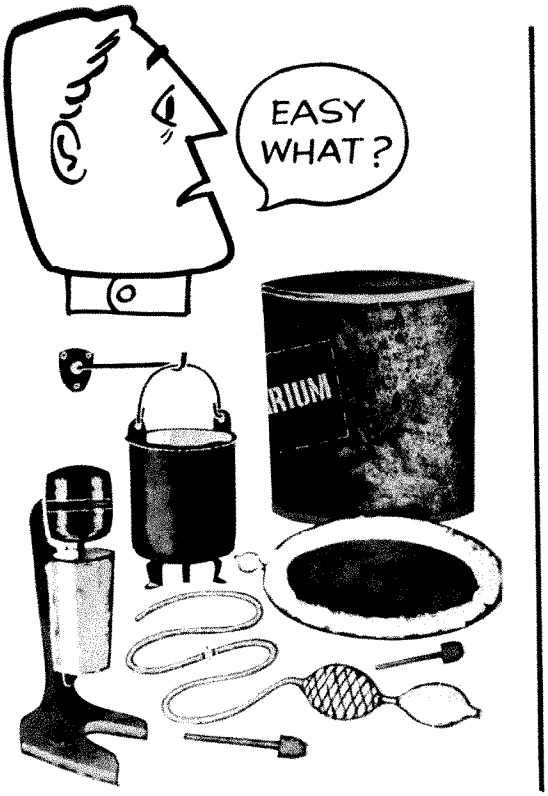
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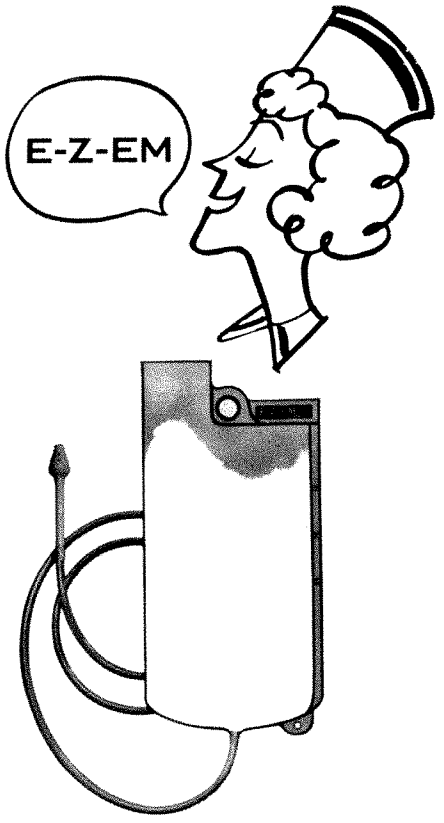
The extremely large vertical travel of the spot film (40"), together with motor-driven table top travel, generously meets all requirements of examinations, standing or prone. Footrest is moved from one side by adjusting one knob only.

The spot film device is available either hand-operated or motor driven. Both work automatically—without program selector.

Our systems engineers are available to explain the Diagnost 50—and other Norelco models—in detail. Write or call for further information. North American Philips Company, Inc., *Medical Division*, 100 East 42nd Street, New York, New York 10017. In the Chicago area: 749 Howard St., Evanston, Ill.

Norelco®





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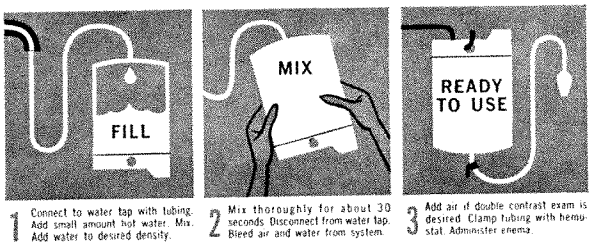
The Single-Stage Double-Contrast Examination, the newest, most convenient, double contrast technique, was developed with, and is based on, the use of E-Z-EM Barium Enema Kits.

Different barium formulations (with or without tannic acid), based on your needs and requests, are available. You choose your technique, and then standardize on it with E-Z-EM. E-Z-EM Barium Enema Bags, without barium, are also available.

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1 Connect to water tap with tubing. Add small amount hot water. Mix. Add water to desired density.

2 Mix thoroughly for about 30 seconds. Disconnect from water tap. Bleed air and water from system.

3 Add air if double contrast exam is desired. Clamp tubing with hemostat. Administer enema.

Complete prepackaged disposable barium colon enema

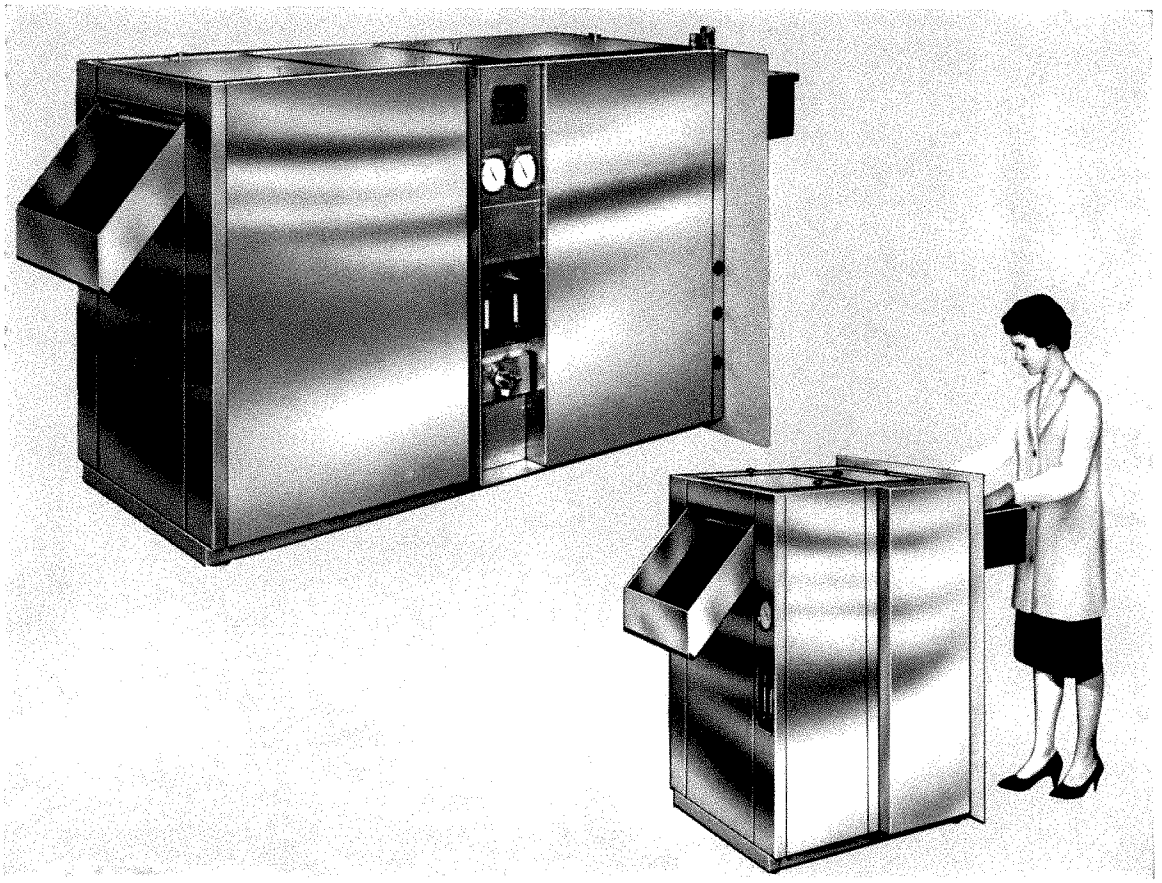
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The PAKOROL-X will process 120, 14″ x 17″ radiographs per hour —the PAKOROL-XM processes 65 14″ x 17″ radiographs per hour—all with uniform quality.

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The Pakorol-XM (miniature) is so small, neat and economical, multiple installations have often proven to be the answer to efficiency, speed and cost cutting.

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Some of the many reasons the Pakorol-X and XM drew crowds at recent conventions are: Full worm and gear driven racks, no chains; quiet roller type operation; 6½ minutes processing time (Pakorol-X) 7 minutes (Pakorol-XM) Right and Left hand models; large capacity; processes all brands of medical screen film in sizes 4″ x 4″ through 17″ x 36″ and roll film in widths of 17″ or less; No. 4 satin stainless steel finish.

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PAKOROL EQUIPMENT is backed by established Pako Parts and Service programs. User training courses are offered regularly at the Pako factory. Let the Pako Planning Service show how the Pakorol-X and the Pakorol-XM will fit your X-ray Department. A suggested layout, tailored to your requirements and space will be prepared upon request, at no obligation.

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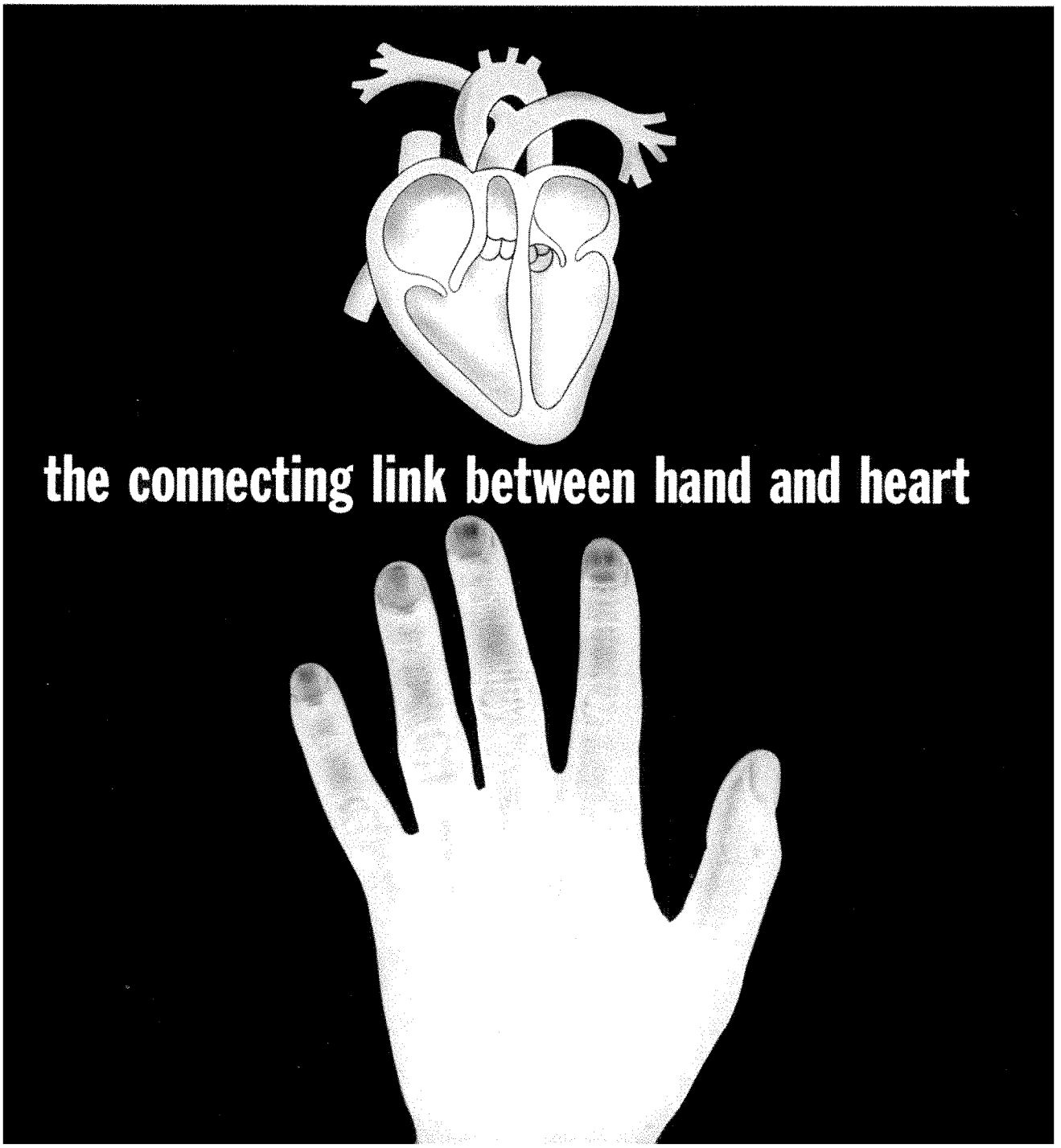
inecting the gap between the surgeon's hand and patient's heart are B-D Arterial Catheterization fits; skillfully engineered and formed of the finest terials. Each contains a thin-wall arterial needle, exible guide, radiopaque tubing in small, medium, arge size, and a LUER-LOK adapter.

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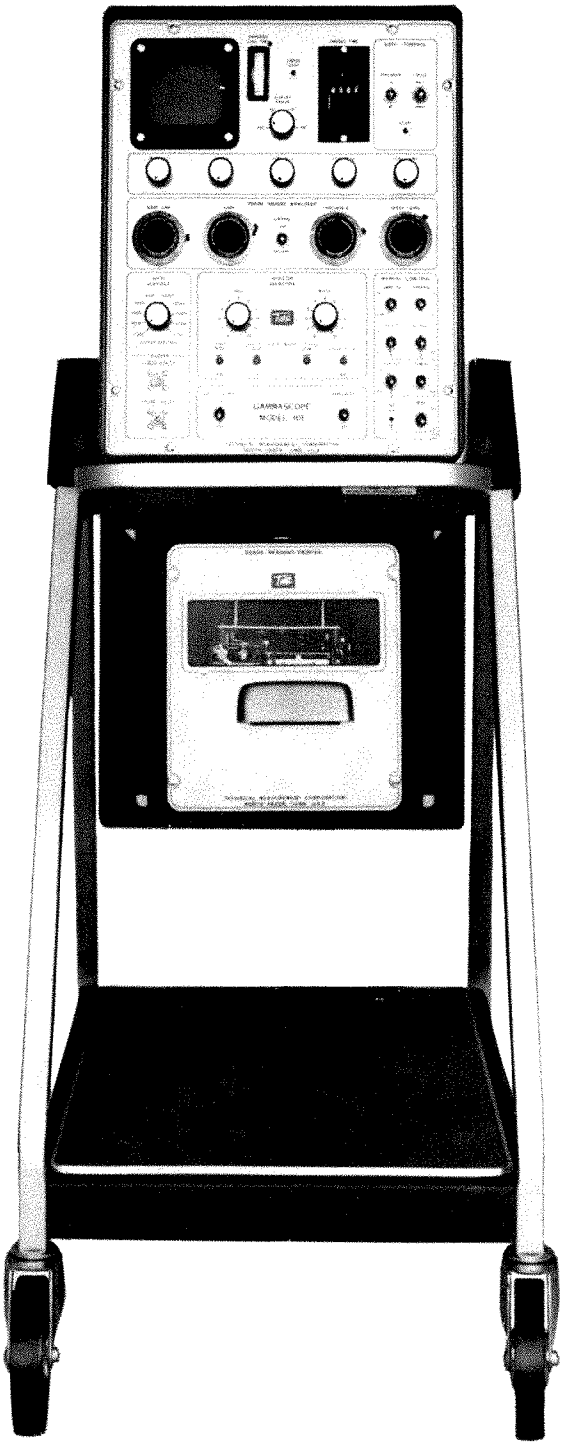
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Half-lives of less than a day are problems for scanning devices, but the Gammascope, with fast automatic data accumulation, can complete a spectrum analysis in far less time than it takes to materially affect the isotope's activity.

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For complete data contact the nearest TMC office or Technical Measurement Corporation, 443 Washington Avenue, North Haven, Connecticut.

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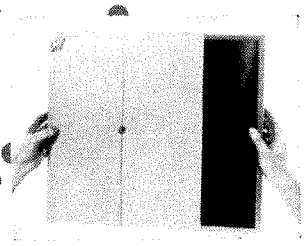
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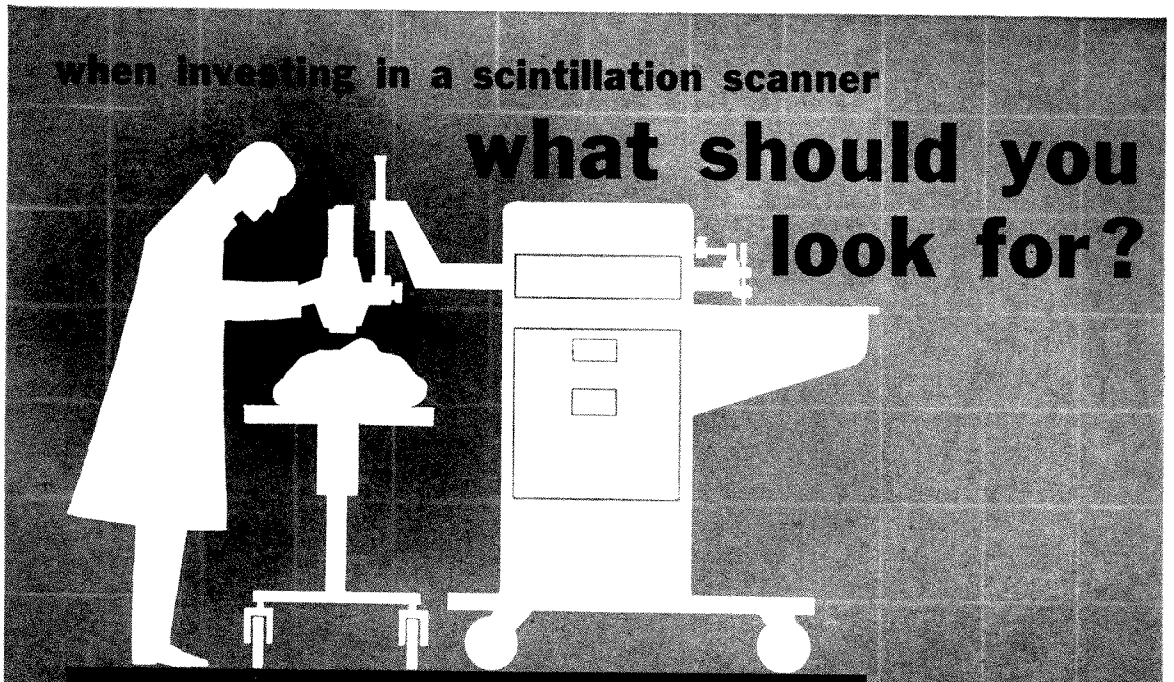
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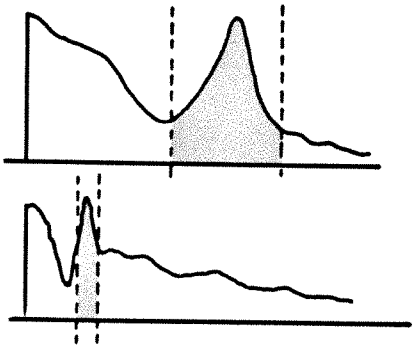
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Set the window anywhere in the spectrum: make it as narrow or as wide as you want.

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The Picker Magnascanner lets you set the window anywhere in the spectrum and lets you make it as narrow or as wide as you want. Some other scanners restrict choice to one of three fixed windows.

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From the contents of Vol. 33, No. 2

BURHENNE, H. J. (San Francisco, Calif.): Intestinal Evacuation Study: A New Roentgenologic Technique

REUTER, S. R. and NEWTON, T. H. (San Francisco, Calif.): Bilateral Atherosclerotic Occlusion of the Common Carotid Arteries

Future issues will include:

BLOEDORN, F. G. and WIZENBERG, M. J. (Baltimore, Md.): Preoperative Irradiation

PIRK, F. and VULTERINOVA, M. (Prague): The X-Ray Picture of the Small Intestine and Impaired Absorption

PALUBINSKAS, A. J.; PERLOFF, D. and WYLIE, E. J. (San Francisco, Calif.): Curable Hypertension due to Renal Artery Lesions

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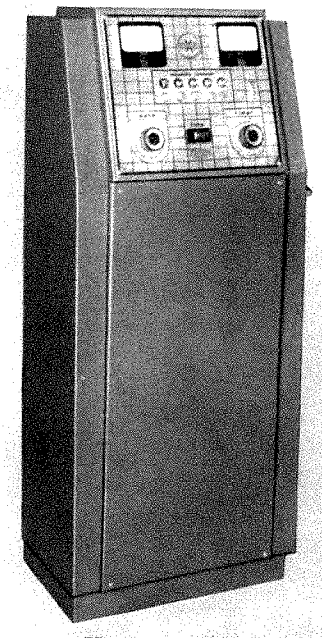
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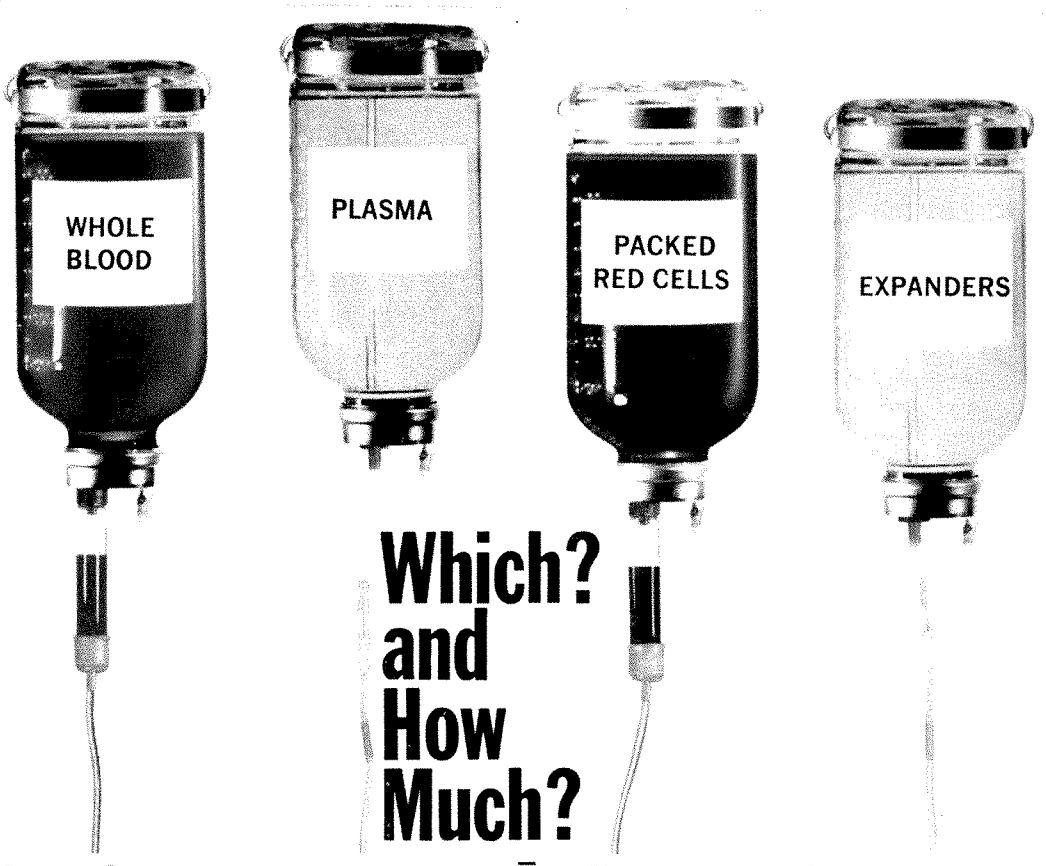
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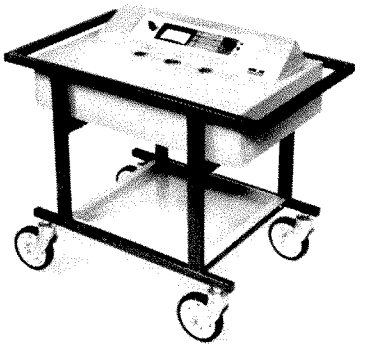


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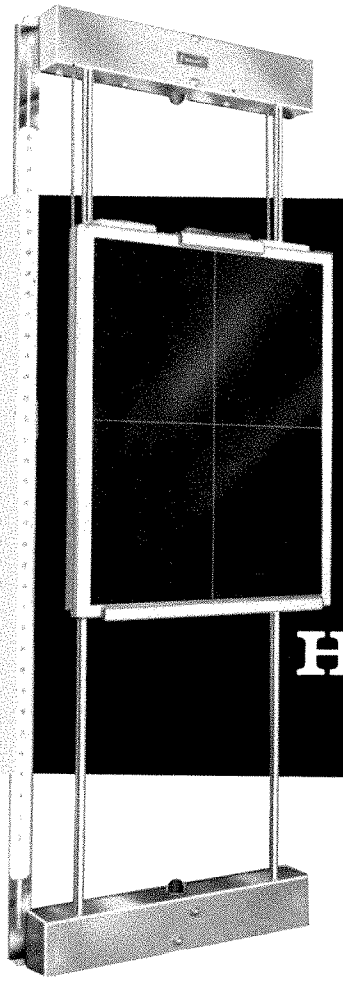
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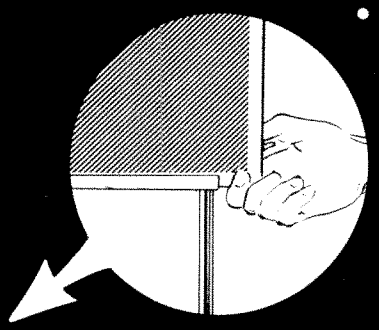
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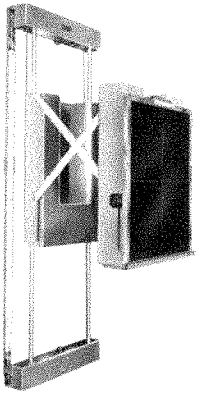
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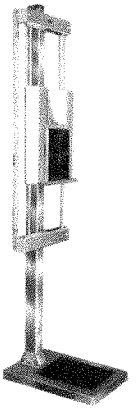
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LYMPHATIC DYNAMICS IN CERTAIN ABNORMAL STATES*

By S. WALLACE,† L. JACKSON,‡ G. D. DODD, *and* R. R. GREENING
PHILADELPHIA, PENNSYLVANIA

LYMPHANGIOGRAPHY is now a useful clinical method. Primary emphasis has been placed upon gross morphology with relatively little attention directed toward the dynamics of lymphatic flow. However, as a "third" component of the vascular system, the fundamental principles of hemodynamics are applicable to the circulating lymph. The physical forces governing the fluid media in the lymphatics, arteries, and veins are intimately related to one another and to the interstitial fluid pool; abnormalities in any one of these compartments will be mirrored by changes in the others in an attempt to maintain equilibrium. Although at present only the skeletal lymphatics can be examined with any degree of constancy, certain abnormal stages so alter intralymphatic directional flow that information relative to the visceral lymphatics and collateral anastomotic channels is obtainable. While relatively complex techniques, such as cineroentgenography, are of value in the study of lymphatic dynamics, the velocity of flow in most channels is negligible as compared with the venous or arterial systems, and, for this reason, abnormal flow patterns may be identified and followed by conventional roentgenographic techniques. Numerous departures from the accepted norm may be observed and anastomotic channels, heretofore only suspected, may be identified. The effect of these upon directional lymphatic flow in the presence of obstructive phenomena has cast considerable doubt upon the efficacy of certain procedures now considered an integral part of cancer surgery. While lymphangiography is in its infancy and our data are fragmentary, we feel that sufficient information has been gained to provide a basis for future investigation.

MECHANICAL OBSTRUCTION

As is the case with arteries and veins, mechanical obstruction of lymphatics results in varying degrees of utilization of available collateral channels. Usually, continuation of lymphatic flow is accomplished

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† National Institute of Health Postdoctoral Fellow.
‡ Leukemia Society Fellow.

by those channels most immediately adjacent to the site of interruption. In its simplest form, this is illustrated in Figure 1*A*, a patient with a healed surgical incision of the lower extremity. The lymphangiogram clearly demonstrates a network of collateral channels at the site of the incision. In this instance, the dermal lymphatics serve as convenient anastomotic channels which re-establish continuity of flow. Utilization of a second collateral pathway is shown in Figure 1*B*. Normally, the lymphatics of the anterior compartment of the lower extremity are functionally separate from those of the posterior compartment. In this instance, complete obstruction of the superficial lymphatic trunk in the anterior compartment has resulted in opacification of the deeper posterior channels via the dermal and subcutaneous networks. The double chain of posterior lymphatics lies in close approximation to the deep arteries and drains into the popliteal lymph nodes.

Following lymph node dissection, there is interruption of numerous lymphatic channels. In order to maintain lymph flow, extensive collateral circulation must be established to compensate for this obstruction. If the attempt to circumvent the block is inadequate, the system decompensates and edema results. This series of events was noted in a patient examined following a right inguinal lymph node dissection for metastatic melanoma from a primary lesion of the skin of the right lower extremity (Fig. 2, *A*, *B* and *C*). Shortly after surgery, the patient noted progressive edema of the extremity. A lymphangiogram demonstrated multiple collateral channels in the right leg, primarily in the

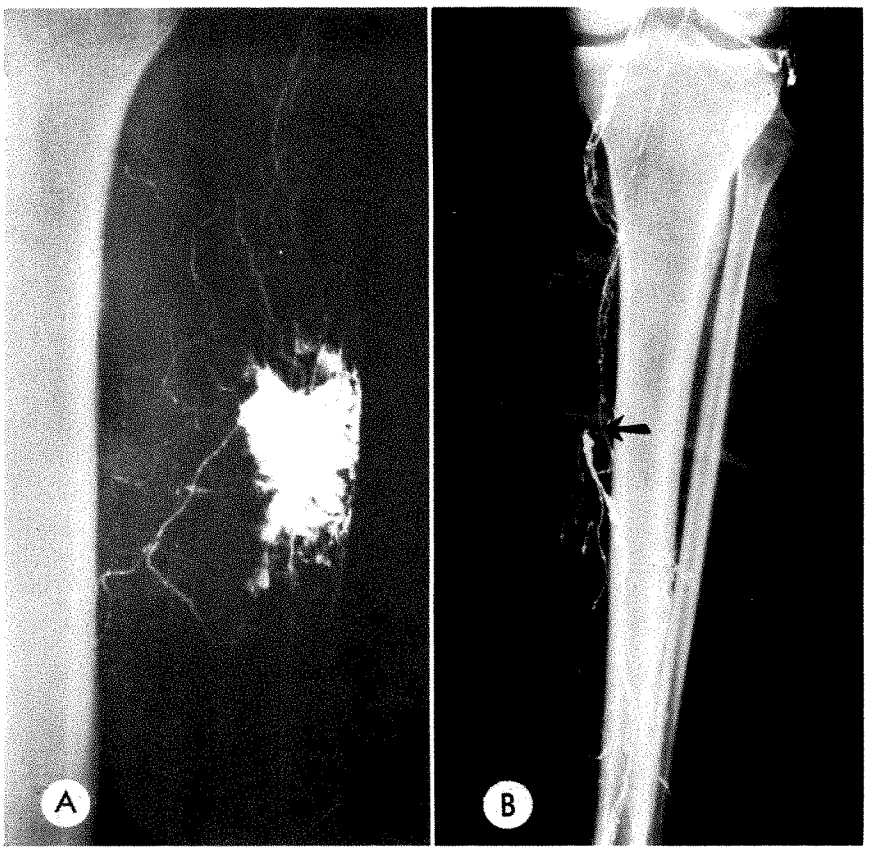


FIG. 1. *Mechanical obstruction with collateral circulation.* (*A*) Collateral circulation at the site of a skin incision. (*B*) Obstruction of the anterior lymphatic trunk (arrow) with collateral channels paralleling the arteries. (Busch and Sayegh, U. S. Naval Hospital, Philadelphia, Pennsylvania.)

dermal and subcutaneous lymphatic networks. Subsequent 24 hour lymphangiograms revealed opacification of the lymph nodes in the right posterolateral aspect of the abdomen in contrast to the normal para-aortic distribution seen on the left. This alternate route portrays another potential compensatory mechanism by which interference with lymphatic circulation is handled. In this instance, however, the subsequent edema is clinical evidence of the inadequacy of the collateral pathways.

The potential collateral lymphatic pathways are complex and extensive. The patterns are not predictable but the diversity indicates the futility of radical dissection in the presence of lymphatic obstruction. This was observed on lymphangiograms of patients with obstruction of lymphatic channels in the pelvis, retroperitoneal area, and the thoracic duct. Both skeletal and visceral channels are utilized, depending upon the accessibility and patency of these collateral routes (Fig. 3, *A–D;* and 4, *A–E*).

This diversity has been further demonstrated by Wohlgemuth[63] of the University of Maryland in a patient with a carcinoma of the cervix and 2 large firm lymph nodes in the left axilla. Lymphangiography was performed to determine the extent of pelvic metastases. This revealed bilateral obstruction in the pelvis with visualization of the internal iliac pathways, a secondary sign of metastasis to the external iliac chain. In addition, a single channel coursed along the anterior abdominal and chest walls to the left axilla. Metastasis to the axillary lymph nodes had actually occurred via this collateral as a sequel to the widespread pelvic disease. This case shows the complexity and unpredictability of metastatic patterns in the face of obstruction.

In general, it may be stated that the establishment of collateral circulation depends upon the available adjacent lymphatic channels; *i.e.*, a block in any portion of the lymphatic system may result in establishment of collateral channels at some distance, since these may represent the only available bypass. The obstruction produces an intralymphatic hypertension which is relieved or partially relieved by the first available avenue. This is shown on Figure 5, *A* and *B*, a patient who was examined because of a lesion in the posterior mediastinum. The lymphangiogram demonstrates obstruction of the thoracic duct at the level of the lesion. Below the point of

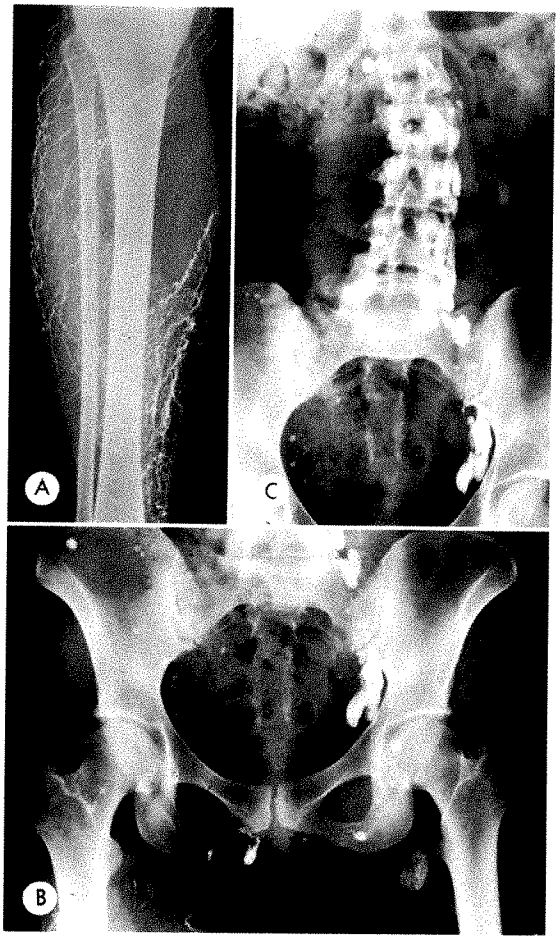


Fig. 2. *Lymphatic obstructions following right inguinal lymph node dissection for metastatic melanoma.* (*A*) Collateral circulation by way of subcutaneous and dermal lymphatics. (*B*) The right inguinal lymph nodes have been removed. The posterolateral pelvic lymph nodes are visualized by way of collateral flow. The left iliac and inguinal lymph nodes are in the usually visualized distribution. (*C*) The posterolateral retroperitoneal lymph nodes visualized via collateral channels can be compared to the normal para-aortic lymph nodes seen on the left.

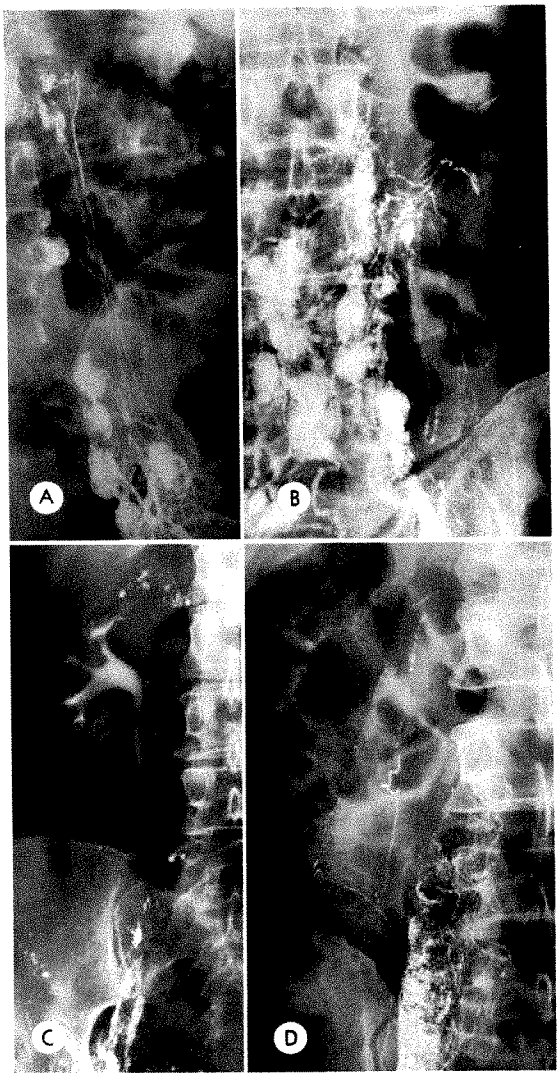


FIG. 3. *Collateral channels in the retroperitoneal area.* (*A*) Abnormal channels are seen in the retroperitoneal area in this patient with Hodgkin's granuloma. (Pick, Albert Einstein Medical Center, Northern Division, Philadelphia, Pennsylvania.) (*B*) Similar changes demonstrated in another patient with Hodgkin's granuloma. (Pick, Albert Einstein Medical Center, Northern Division, Philadelphia, Pennsylvania.) (*C*) Obstruction of pelvic lymphatics by metastatic carcinoma from a primary in the cervix resulted in the visualization of the perivascular lymphatics in the pelvis. Another collateral pathway is filled in the retroperitoneal area. (*D*) Lymphatic vessels along the psoas muscle function as alternate channels in this patient whose lymph nodes are involved by Hodgkin's disease.

interruption, there is dilatation of the abdominal portion of the thoracic duct and the cisterna chyli with visualization of collateral pathways into the abdomen. Thus the primary alternate route was via the gastrointestinal trunk, which represented the first available avenue of decompression.

While the identification of collateral pathways may frequently be of value in the detection of lymph node replacement by tumor tissue, the number and distribution of the normal lymphatic channels may vary greatly. The lymphatic vessels function primarily as a vascular network and may, under normal circumstances, bypass or exist independently of the lymph nodes. In such instances, the demonstration of single or multiple channels in the normal paraaortic distribution does not necessarily infer that the vessels represent collaterals or that total replacement of the lymph nodes has occurred. The patient demonstrated in Figure 6*A* suffered from carcinoma of the breast with widespread metastasis and ascites. Obstruction of the cysterna chyli was postulated and lymphangiography was performed for confirmatory purposes. The total absence of lymph nodes was felt to represent extensive metastatic disease, but at autopsy an absence of the retroperitoneal lymph nodes was found. This then is a congenital variant. Similarly, following radiation therapy or chemotherapy, there may be a decrease in size or complete disappearance of the lymph nodes as seen in Figure 6*B*. This patient had Hodgkin's disease and was treated by both modalities. More frequently, as lymph node replacement occurs, only occasional lymphatic channels may be visualized with the deposition of contrast material in few or none of the regional lymph nodes. The number of lymphatic channels may vary greatly but they are characterized by flow of the contrast medium along the normal lymphatic distribution. The patient in Figure 6, *C* and *D* had known Hodgkin's disease and characteristic lymphomatous lymph

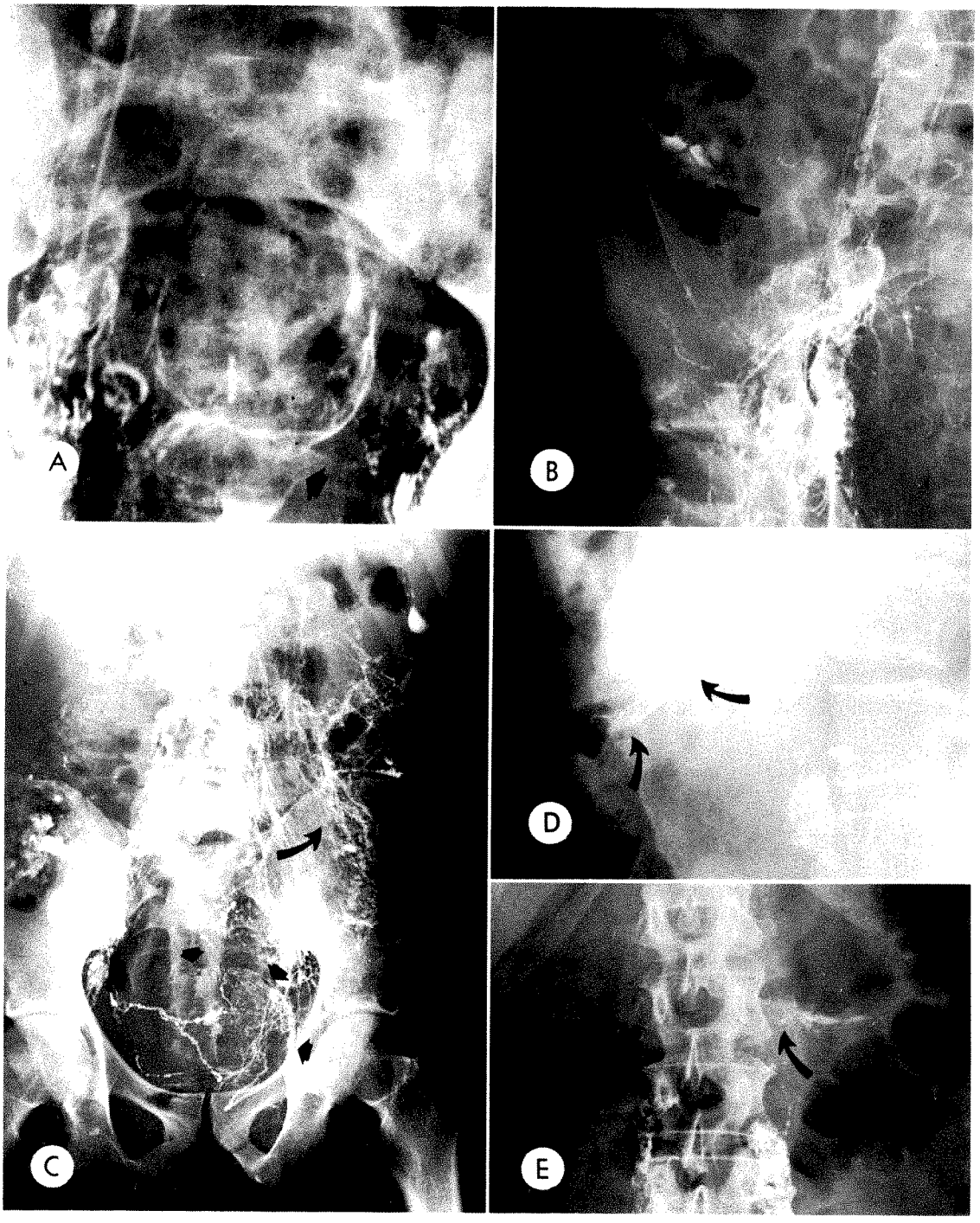


Fig. 4. *Alternative pathways via the intestinal lymphatics.* (*A*) Obstruction of the pelvic lymphatics resulted in visualization of the channels around the rectum (short arrows). (*B*) Collateral channels eventuated in the demonstration of the mucosa of the bowel (long arrow). (*C*) Interruption of lymphatic trunks at the level of the cisterna chyli produced reversal of flow into the lymphatics of the bowel (long arrow) and bladder (short arrows). (M. D. Anderson Hospital.) (*D* and *E*) An attempt to circumvent mechanical obstruction of the lymphatics is seen via the lymphatics of the stomach (long arrows).

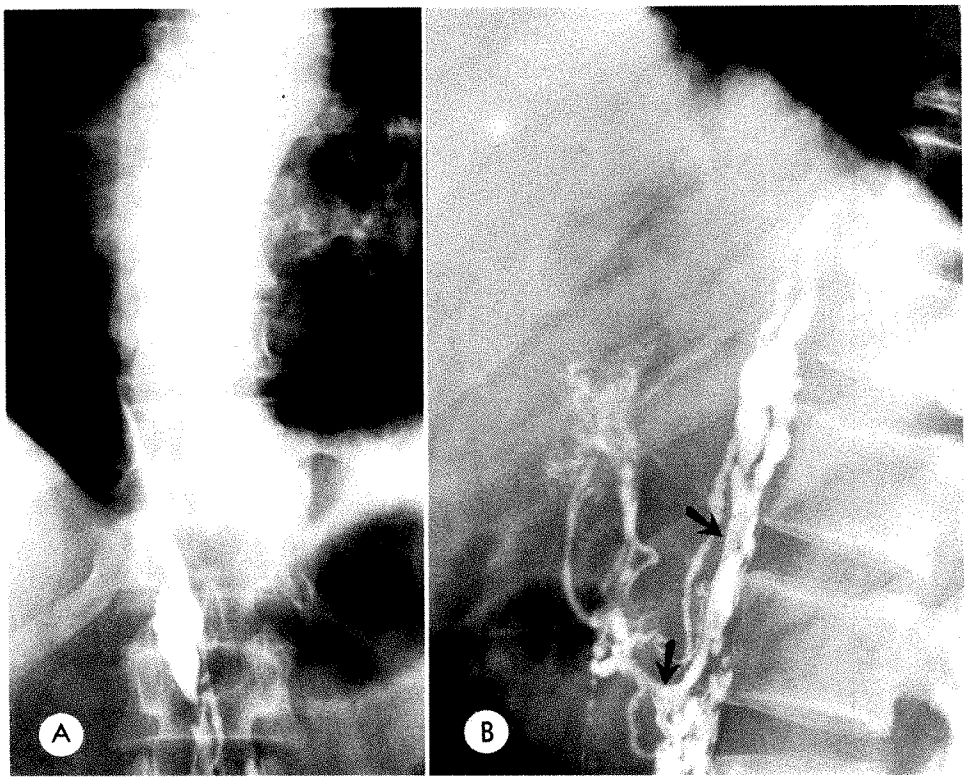


FIG. 5. *Obstruction of the thoracic duct.* (*A*) A posterior mediastinal mass obstructed the thoracic duct producing dilatation of the cisterna chyli. (*B*) Retrograde flow is seen into the gastrointestinal trunk (arrows). The major channels forming the cisterna chyli, the right and left lumbar trunks and the gastrointestinal trunk are readily identified.

nodes in the pelvis were demonstrated on the lymphangiogram. The retroperitoneal area was devoid of lymph nodes but a few channels traversed the usual course of the para-aortic plexus. The interpretation of such findings is difficult. For this reason, inferior vena cavagraphy is frequently performed in an attempt to separate normal variants from total lymph node replacement. The inferior vena cavagram in this case confirmed the presence of massive lymph node replacement which indented and distorted the contrast filled vessel. Therefore, the addition of inferior vena cavagraphy is frequently essential in fully evaluating or appreciating changes of this type.

CHYLOUS REFLUX

Probably the most dramatic example of alteration of the normal lymphatic flow pattern by obstruction is chylous reflux. This, as the name implies, represents a reflux of chyle from the intestinal lymphatics into the skeletal lymphatics. This phenomenon may result from either congenital or acquired abnormalities of the lymphatic system. The usual congenital abnormality is a primary incompetence of the lymphatic valves with stasis as a result of failure of directional flow. The acquired type may be associated with any process which produces chronic thoracic duct obstruction, with filariasis probably the most common agent. The dilatation of the lymphatics distal to this point of obstruction results in a secondary incompetence of the lymphatic valves, producing a picture which is clinically identical with the congenital variety.

These patients usually complain of progressive edema of the extremities and chyluria. Abeshouse in 1934 described

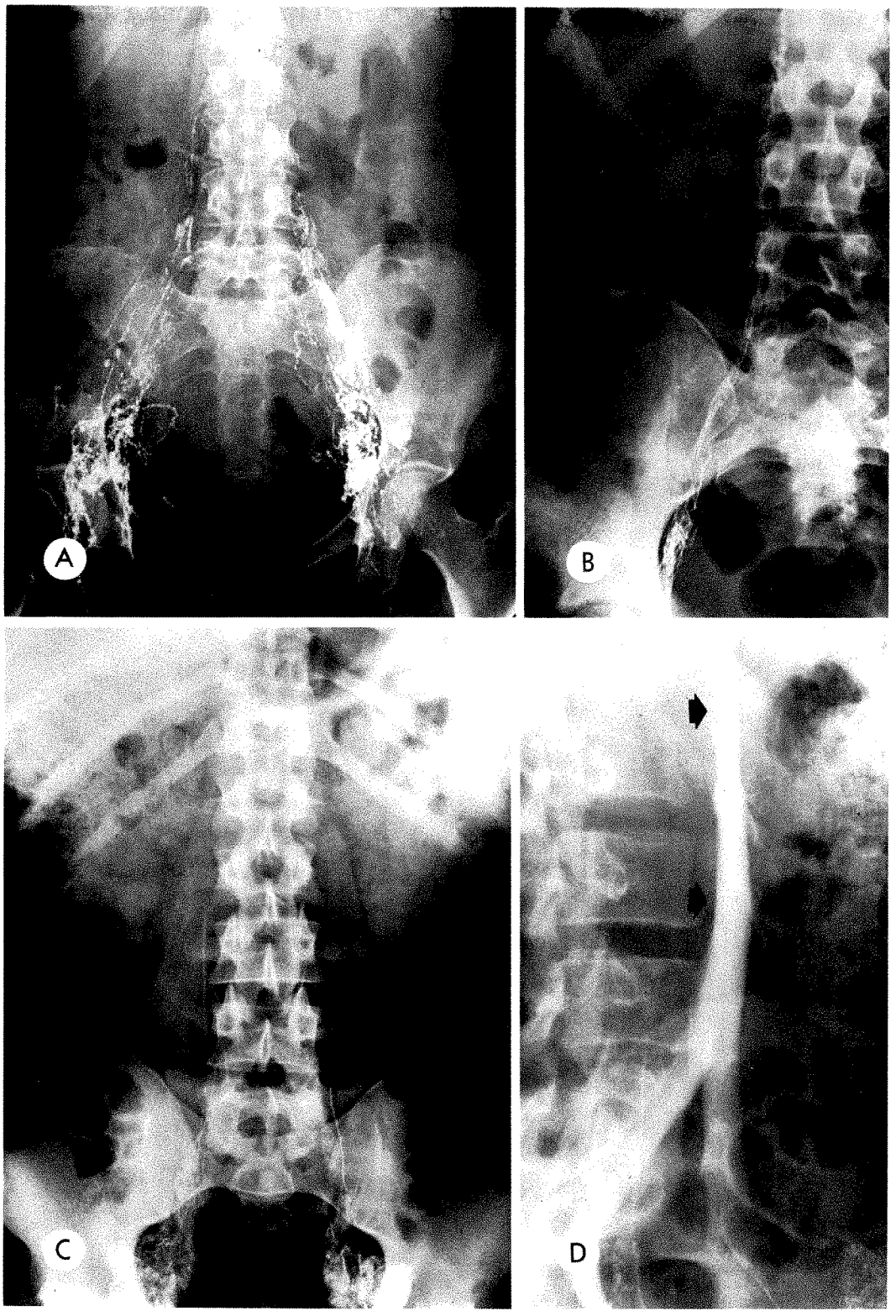


FIG. 6. (*A*) Absence of retroperitoneal lymph nodes in a patient with carcinoma of the breast proved to be a congenital variant. (*B*) This patient with Hodgkin's granuloma was treated by radiation therapy and chemotherapy with a resultant paucity of lymph nodes. (*C*) The failure of visualization of retroperitoneal lymph nodes in this patient was secondary to total replacement of the lymph nodes with lymphoma. (*D*) A venogram of the patient in *C* confirms the presence of a retroperitoneal mass compressing the inferior vena cava (arrows).

chyluria as a result of retrograde flow of the lymphatics of the abdominal wall where the anastomoses with vessels of the upper half of the body occur. One or more of these collateral vessels may rupture into the pelvis of the kidney, the ureter, or the urinary bladder, giving rise to chyluria. Servelle and Deysson[49] have reported on 6 cases of chylous reflux out of 50 patients with elephantiasis. They found the lymphatic channels enlarged in diameter, ranging from 1 mm. to 1 cm. Retrograde flow was believed due to the incompetency of the valves which resulted from marked distention of the obstructed lymphatics. They felt that the initial process was one of an acute adenitis with subsequent sclerosis of the lymph nodes producing lymphatic obstruction. This, in turn, resulted in lymph stasis, dilated lymphatics, and eventual edema. In one of their patients, there was a coexistent congenital malformation of the veins. Kinmonth and his co-workers[29,30,31] and then Jantet[26] summarized the total of 9 cases of chylous reflux reported in the literature. It was their belief that this was a rare complication of primary lymphedema with retrograde flow directed along the posterior abdominal wall to the pelvis and lower extremities. They noted that vesicles were frequently seen in the skin of the genitalia and lower extremity, which, when punctured, would exude intestinal chyle.

We feel that the clinical manifestations depend on the site of the involvement and the anatomic variation of the lymphatic system in the individual patient. The anatomic variations primarily occur in the formation of the cisterna chyli from its major components, the right and left lumbar trunks and the gastrointestinal trunk. The classic picture of cisterna chyli as a bulbous dilatation at the junction of three major trunks is seen in approximately 47 per cent of cases.[44,51] However, in a significant percentage, the gastrointestinal trunk drains into the major lumbar lymphatic channels, either as a single trunk or multiple smaller ones. This would explain the presence of unilateral chylous edema of the extremity or unilateral chyluria. Unilateral findings have been more frequently noted on the left.

A patient who exemplified this phenomenon was studied by C. M. Henner, Methodist Hospital, Dallas, Texas, because of chyluria and chylous edema of the stump of the lower left extremity which had been amputated years before (Fig. 7, *A–D*). An extensive network of lymphatics was seen following the cannulation and injection of a superficial lymphatic in the right foot. The pathways clearly showed the multidirectional flow. Intercostal lymphatics were filled and were seen to terminate in the visualized axillary lymph nodes. The presence of contrast medium in the pelvocalyceal system was an indication to the mechanism involved in the production of chyluria. The lymphatic channels in the pelvis were markedly dilated and tortuous and reflux was demonstrated in the stump of the opposite extremity and scrotum. It is of some interest that scattered collections of radiopaque material found in vesicles in the skin of the scrotum were similar to those described by Kinmonth *et al.* and Jantet.

In addition to the described forms of chylous reflux, most of which are of a chronic nature, it is apparently possible to produce a temporary chylous reflux by lymphangiography. The major portion of the lymph is gathered in the lacteals of the small intestine with an almost negligible amount generated in the normal lower extremities. The intestinal chyle passes centrally to the aortic lymph nodes en route to the thoracic duct. Although collateral pathways exist, the primary flow is via the retroperitoneal plexus. During the performance of lymphangiography, a large amount of insoluble oil based medium enters the lymph nodes. This can apparently produce a temporary stasis of lymphatic flow due to simple congestion of the lymph node and an increase in the pressure gradient within the skeletal pathways due to the presence of the more viscid contrast material. This was noted in a patient in

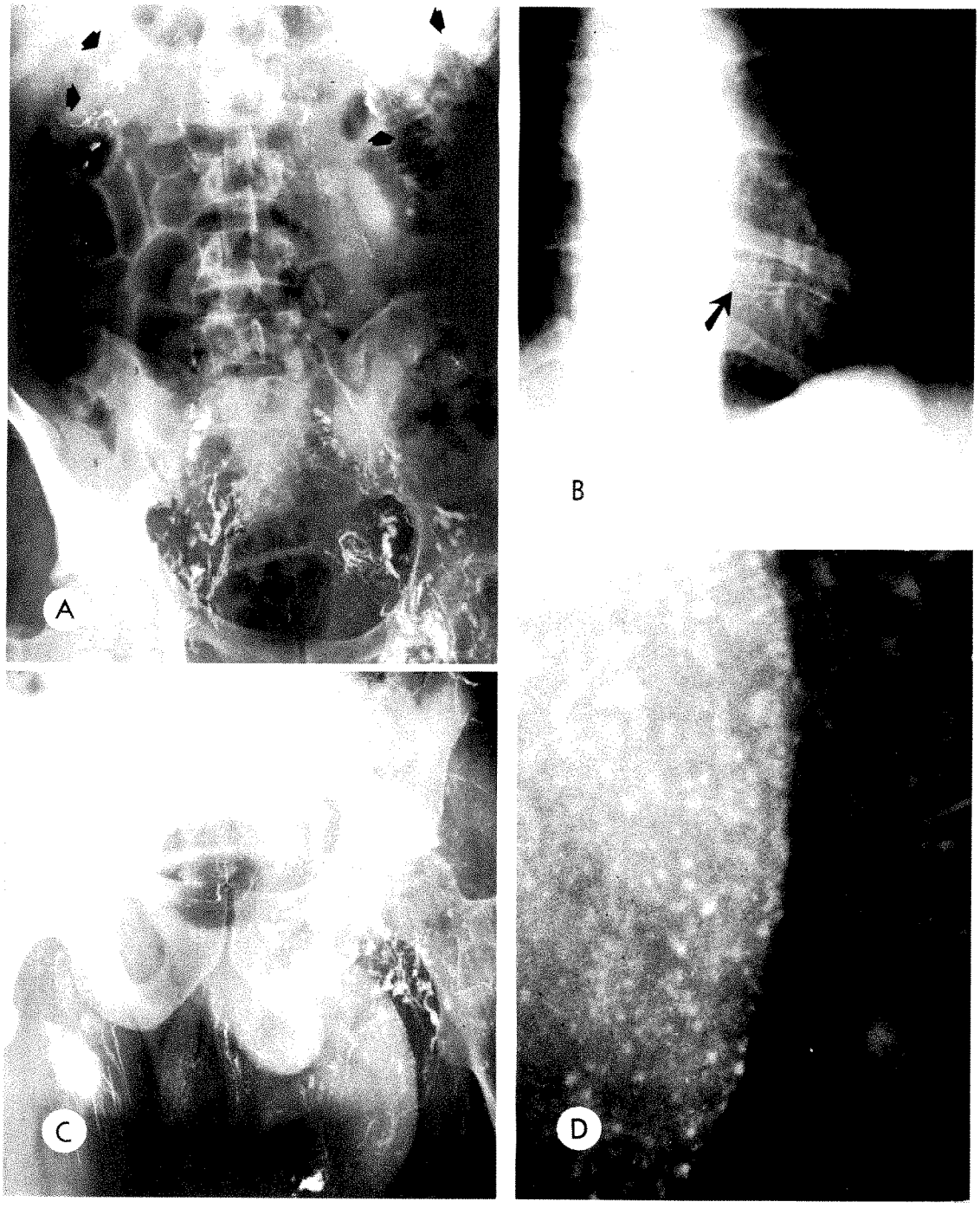


Fig. 7. *Chylous reflux.* (*A*) The dynamics of chyluria are demonstrated by the visualization of the pelvocalyceal systems of each kidney (arrows). (*B*) Reflux into the intercostal lymphatics (arrow) resulted in the demonstration of the left axillary lymph nodes. (*C*) Retrograde flow into dilated tortuous lymphatic vessels of the pelvis, scrotum, and left lower extremity is seen. (*D*) Contrast filled vesicles are demonstrated in the scrotum. (C. M. Henner, Methodist Hospital, Dallas, Texas.)

our series who was operated on one day following lymphangiography. The lymphatics of both the small and large bowel were dilated and engorged by white intestinal chyle which is ordinarily not present in the large bowel lymph channels. This does not produce a permanent interference with the normal flow pattern, but does have a suggestive usefulness in aiding the surgical identification of mesenteric lymph nodes and channels, particularly when vital dyes are used in conjunction with the oil based media.

NEUROVASCULAR LYMPHATIC SPACES

Thus far, the discussion has been concerned with the utilization of collateral and anastomotic channels which are unequivocally part of the lymphatic system. Recently, we have successfully demonstrated an additional collateral or anastomotic route which we feel is functionally, if not phylogenetically, a part of the lymphatic system. These accessory pathways follow the major vessels, forming a sheath-like structure with an actual or potential space between the vessel wall and the enveloping membrane. Whether or not they constitute a part of the lymphatic system is open to question; however, they are indisputably in communication with the lymphatic vessels. In this respect, they are analogous to the perineural and endoneural spaces.[20,38,60] The clinical importance of the latter in the dissemination of tumor has been emphasized by several investigators.[44] Since the neural lymphatics or spaces provide a relatively unopposed pathway along which tumor cells may propagate centrally from a peripheral lesion, it would seem logical that they may be utilized as collateral pathways in the presence of obstruction. The same reasoning may be applied to the perivascular spaces. We have had the opportunity of observing a tubular cuff of contrast material in several patients which clearly followed the distribution of the major vessels. Sequential roentgenograms have indicated that this is not representative of extravasation of the opaque medium and the caliber and distribution of the columns were suggestive of contrast material lying primarily within the perivascular or perineural sheath. Figure 8, *A*, *B* and *C* shows a patient in whom lymphangiography was performed because of edema of

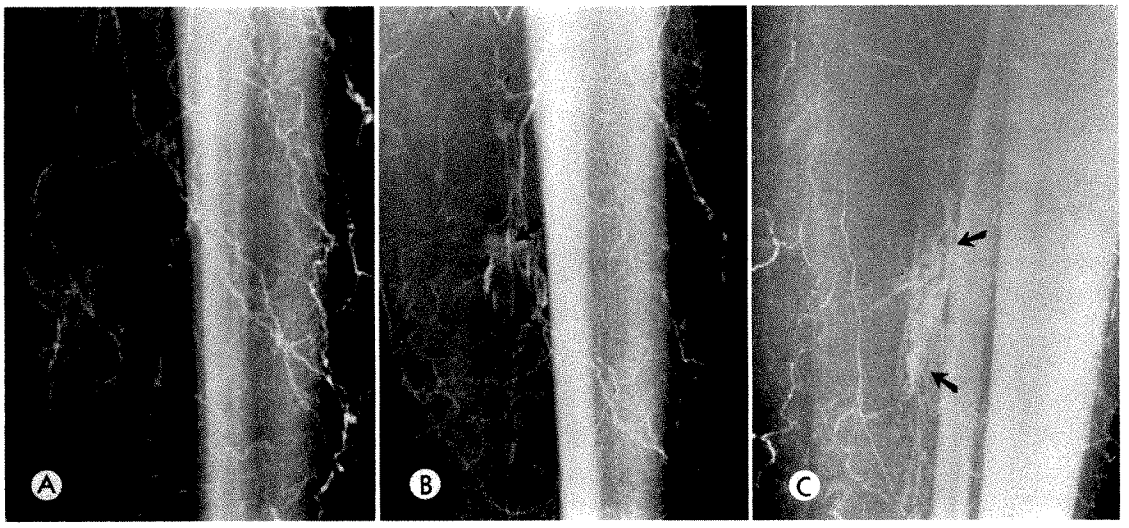


FIG. 8. *Neurovascular lymphatic spaces.* (*A*) An alternative avenue of flow is demonstrated on the first day in the patient studied because of edema of the lower extremity. This initial roentgenogram shows extensive collateral circulation by way of dermal and subcutaneous channels. (*B*) The second day study shows a tubular cuff of contrast material (arrows). (*C*) By the sixth day the cuff was more obvious, and probably lying in the perivascular space (arrows). Note the marked delay in circulation time.

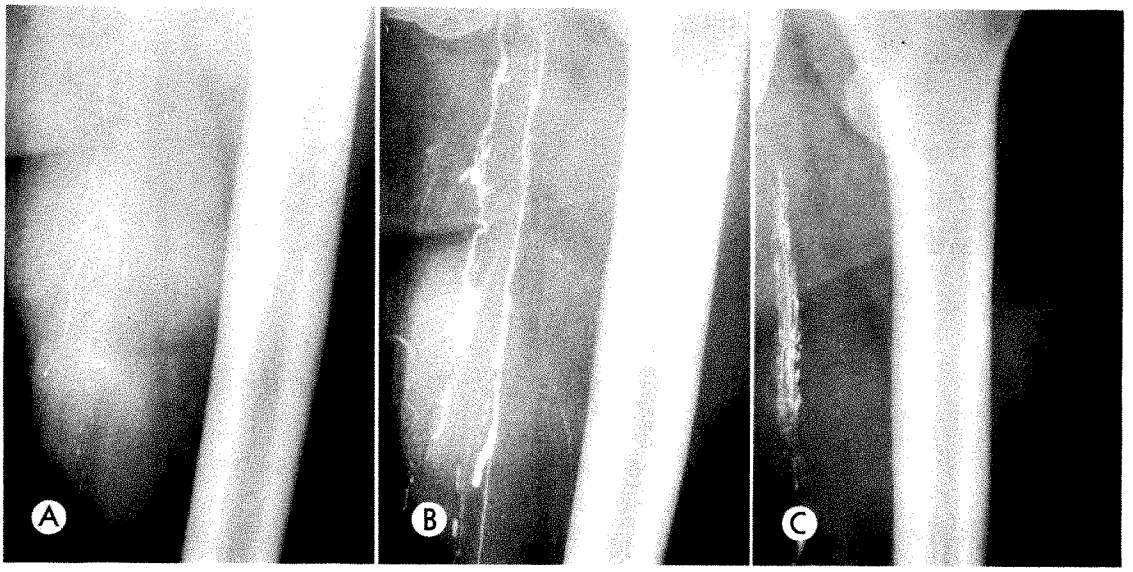


Fig. 9. *Neurovascular lymphatic spaces.* (*A*) Similar findings are noted in the lymphatics of the thigh in the patient described in Figure 8, *A*, *B* and *C*. The initial roentgenogram on the first day reveals little contrast in the lymphatic vessels of the thigh. (*B*) The second day study shows progressive filling of the lymphatics. (*C*) After 6 days a cuff of contrast material is demonstrated.

the lower extremity. Following completion of the injection of 10 cc. of oil based contrast material, a massive lymphatic network was seen. Dermal backflow appeared to be the primary type of collateral circulation. However, on successive days a tubular shadow was outlined which we concluded was within the neurovascular sheath. A similar finding was seen in the thigh as the opaque material progressed proximally (Fig. 9, *A*, *B* and *C*). In other patients, the same configuration was found in the absence of edema. These findings seem to strengthen our hypothesis (Fig. 10, *A* and *B*).

LYMPHATIC-VENOUS ANASTOMOSES

Another channel of flow which is utilized with alterations of lymphatic dynamics is the direct communication between the lymphatics and the veins, the so-called lymphatic-venous anastomoses. These communications have been of considerable interest to anatomists in the past. With the present ability to demonstrate the lymphatics, visualization of such anastomoses becomes important in better understanding lymphatic circulation.

Embryologically, the primary lymphatic sacs bud off from the veins. This in itself creates the potential for such communications. Paired sacs developed from the anterior cardinal veins, forming the jugular sacs which give rise to the major lymphatics of the head, neck, and upper half of the body. A second set of paired sacs develops from the mesonephric vein and these become the iliac sacs, the precursors of the skeletal lymphatics of the lower half of the body. Two single sacs form in common with the iliac sacs, one eventually comprising the anlage for the cisterna chyli and the lower thoracic duct and the other for the lymphatics of the abdominal viscera.

Many investigators[43,44,64] have demonstrated anastomoses between lymphatics and veins other than the communications at the venous angle in the neck. Direct connections exist between the lymphatics and veins of the thyroid, kidney, adrenals, liver, etc. Accessory entrances into the inferior vena cava, azygos, and other veins were visualized after ligation of the thoracic duct by Job,[27] Lee, Blalock *et al.*,[3] Freeman, and others.[44,64] Carlsten and Olin[8] found no such communications initially following li-

gation of the thoracic duct in cats. However, after 2 weeks, flow was established directly into the venous system. Málek *et al.*[36,37] observed in sheep the spread of bacteria from the lymph nodes to the blood stream by way of lymphatic-venous anastomoses. Also when studying anthrax infections by a lymphographic method, Málek *et al.* found direct lymphatic-venous connections at the site of lymph nodes damaged by the inflammation. Pressman *et al.*[39,40] have shown such communication within lymph nodes by the injection of air into the lymph nodes in animals. Air was seen in both the efferent lymphatics and the veins. Frautschi described such communications in human cadavers; however, these are not truly reliable because of the inability to demonstrate lymphatic flow in cadavers. It is possible that fresh entrances into veins may develop under certain circumstances although more likely there is enlargement of pre-existing subsidiary channels which normally carry little or no lymph. The above studies seem to substantiate this supposition.

A recent investigation utilizing direct injection of contrast material into the lymph nodes of living humans disclosed most interesting findings. It was formerly believed that the only communications were at the terminal trunks in the neck. Shanbrom and Zheutlin[50] in 1959 demonstrated accessory lymphatics which drained directly into the jugular vein without traversing a lymph node.

We have seen direct lymphatic-venous communications following intralymphatic injection of contrast material under normal circumstances. This has been most frequently observed in cervical lymphangiography. Also, in the examination of the upper extremities, multiple direct communications between the lymphatics and the subclavian vein other than the classically described junction at the venous angle have been visualized. More frequently, lymphatic-venous anastomoses have been

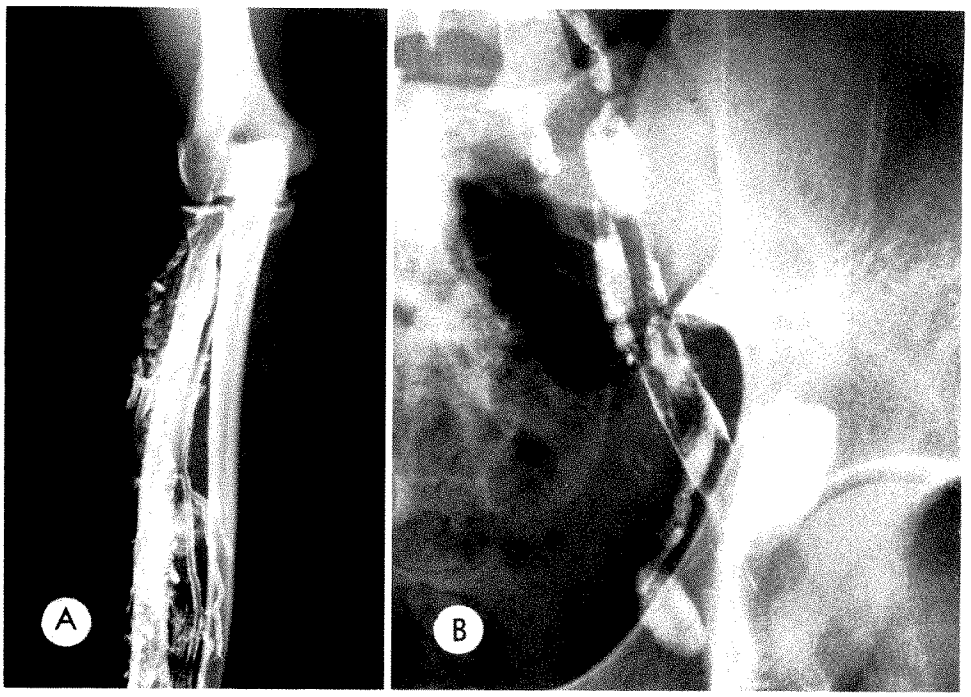


FIG. 10. *Neurovascular lymphatic spaces.* (*A*) The outlining of vascular structure is believed to be due to the lymph flow through perivascular spaces. This patient had no edema. (*B*) Another patient with no edema shows the perivascular lymph spaces visualized by lymphangiography of the lower extremity.

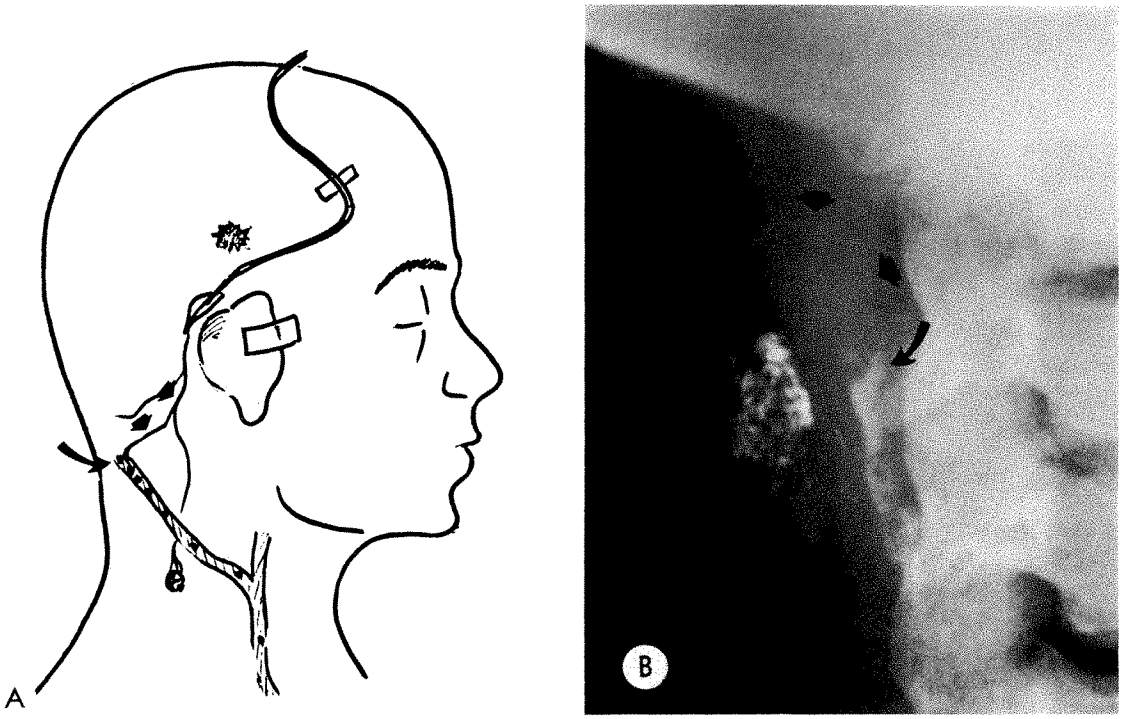


FIG. 11. *Lymphatic-venous anastomosis.* (*A*) Line drawing of the cervical lymphangiogram demonstrates the direct communication of the lymphatics (short arrows) and veins (long arrow) in a patient following a radical neck dissection for malignant melanoma. (*B*) The roentgenogram reveals the irrregular lymph node involved with metastatic disease. A fine lymphatic channel (short arrows) can be seen emptying into a vein (long arrow).

illustrated in patients with obstruction of lymphatic flow, thereby re-establishing the continuity of the system. Gould and Schaffer[17] demonstrated this phenomenon in a patient with carcinoma of the uterus. There was complete replacement of the retroperitoneal lymph nodes by tumor and interruption of the lymphatic pathways. The same mechanism was observed by Wright and Halaby[64] in a patient with a lymphadenectomy and hysterectomy for carcinoma of the cervix. All patients who are examined by lymphangiography will experience pulmonary embolization of the oil-based contrast material. Clinical manifestation of this phenomenon is dependent upon the amount of contrast material entering the lungs and the period of time over which this embolization occurs. In the presence of lymphatic-venous anastomoses, pulmonary embolization is direct and rapid and, therefore, a more serious problem than in the usual examination.

A patient, who was treated 1 year previously by a radical neck dissection for a metastatic melanoma, was examined for the evaluation of a nodule in the operative site. To demonstrate the posterior cervical lymphatics and lymph nodes, the vital blue dye was injected intradermally into the scalp above the ear. A subcutaneous lymphatic was isolated just posterior to the ear. After cannulation and injection of the contrast medium, a channel was demonstrated which terminated in the palpable lymph node which was obviously involved by neoplasm. Another vessel coursed toward the occiput and precipitously emptied its contents into a superficial vein, an example of lymphatic-venous anastomosis (Fig. 11, *A* and *B*). This phenomenon was observed with the use of cineroentgenography. Al-

though the lymphatic remained filled with the radiopaque material throughout its length, the more rapid flow in the vein was manifested by the swift movement of the droplets of contrast material within the venous channel.

Lymphatic-venous anastomoses are probably relatively inactive pathways under normal circumstances. These potential channels are utilized in the event of obstruction to normal flow and are best demonstrated during injection. Once the contrast material enters the vein, it rapidly disappears into the general circulation. When visualized during injection, the examination should be terminated in order to minimize pulmonary embolization of the contrast material.

LYMPHATIC-VENOUS RELATIONSHIP IN EDEMA

Lymphatic dynamics frequently depend on lymphatic-venous interplay. Aside from the direct communication just described, the etiology of many forms of secondary edema lies in the upset of the balance which normally prevails between the two compartments. This is best presented by an analysis of the dynamics associated with the production of edema of an extremity. Differentiation of the mechanisms can be made by performing both lymphangiography and venography. Our findings can be classified as follows: (1) lymphatic obstruction; (2) venous occlusion—(a) intrinsic and (b) extrinsic; and (3) combined lymphatic and venous disease. These classifications are most dramatically illustrated in the variety of mechanisms seen in postmastectomy edema.

1. *Lymphatic Obstruction.* The sequelae of lymphatic obstruction have been described previously in the discussion on collateral circulation. Figure 12*A* shows a case of postmastectomy edema following radical mastectomy and axillary lymph node dissection; the lymphangiographic picture is that usually seen in cases with lymphatic

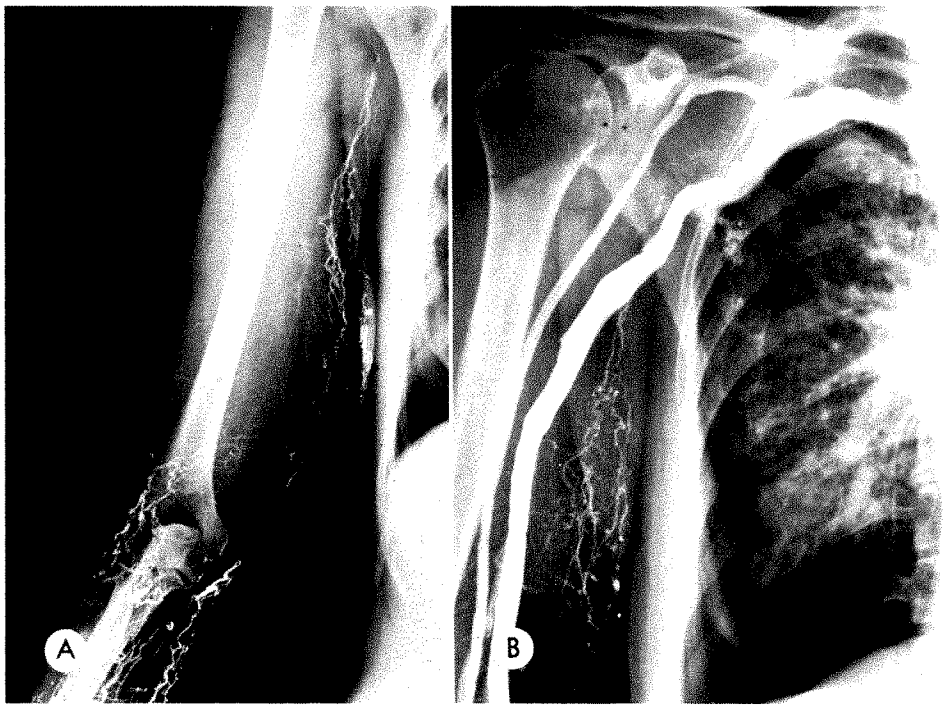


FIG. 12. *Acquired edema of the arm secondary to lymphatic obstruction.* (*A*) Following radical mastectomy this patient developed edema secondary to lymphatic obstruction. (*B*) A venogram of the same patient is normal.

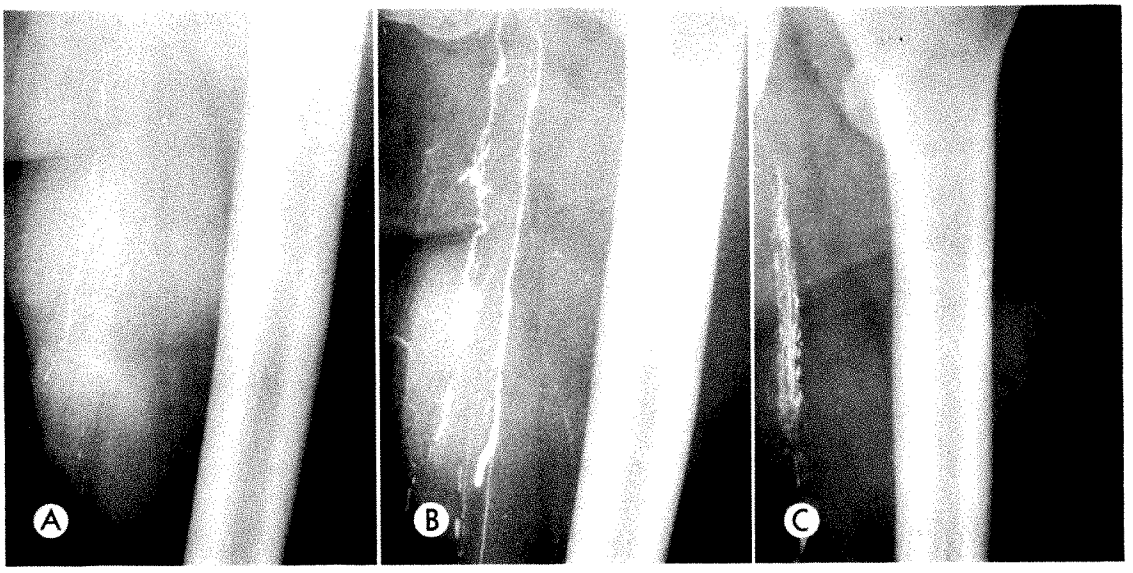


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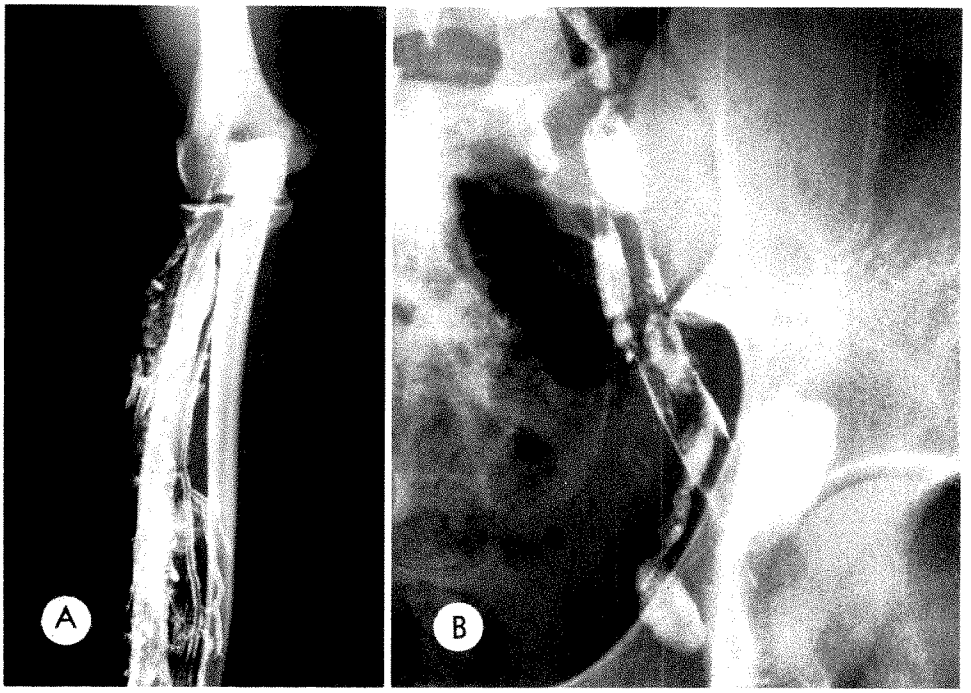


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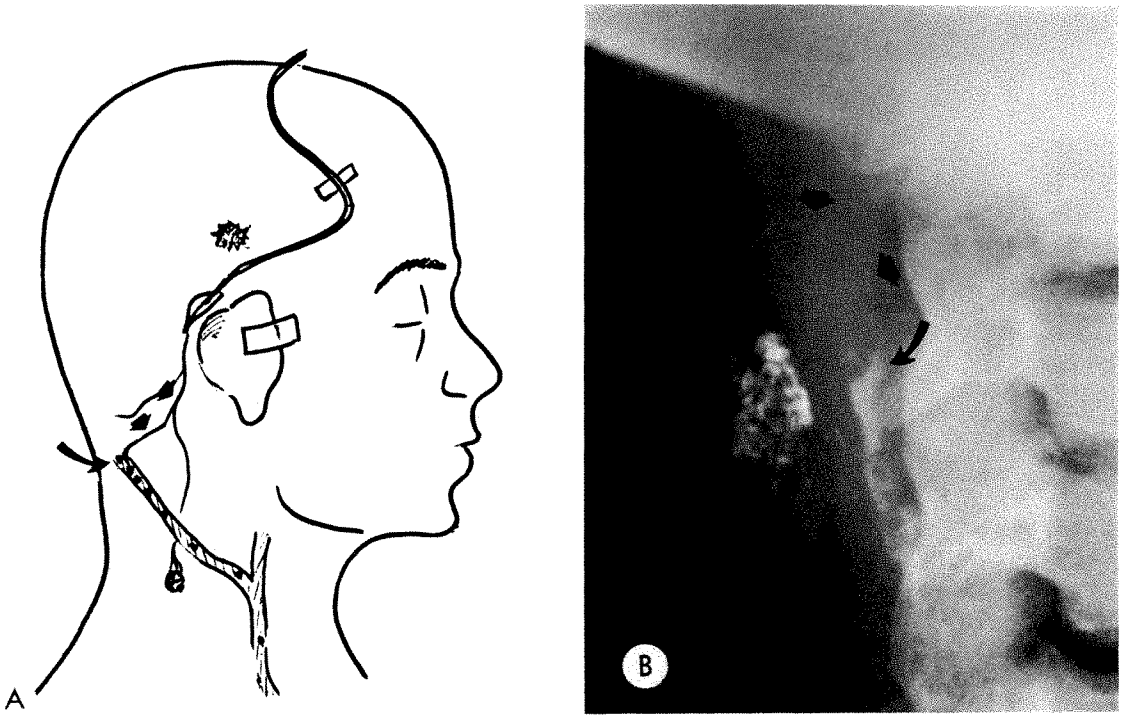


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1. *Lymphatic Obstruction.* The sequelae of lymphatic obstruction have been described previously in the discussion on collateral circulation. Figure 12*A* shows a case of postmastectomy edema following radical mastectomy and axillary lymph node dissection; the lymphangiographic picture is that usually seen in cases with lymphatic

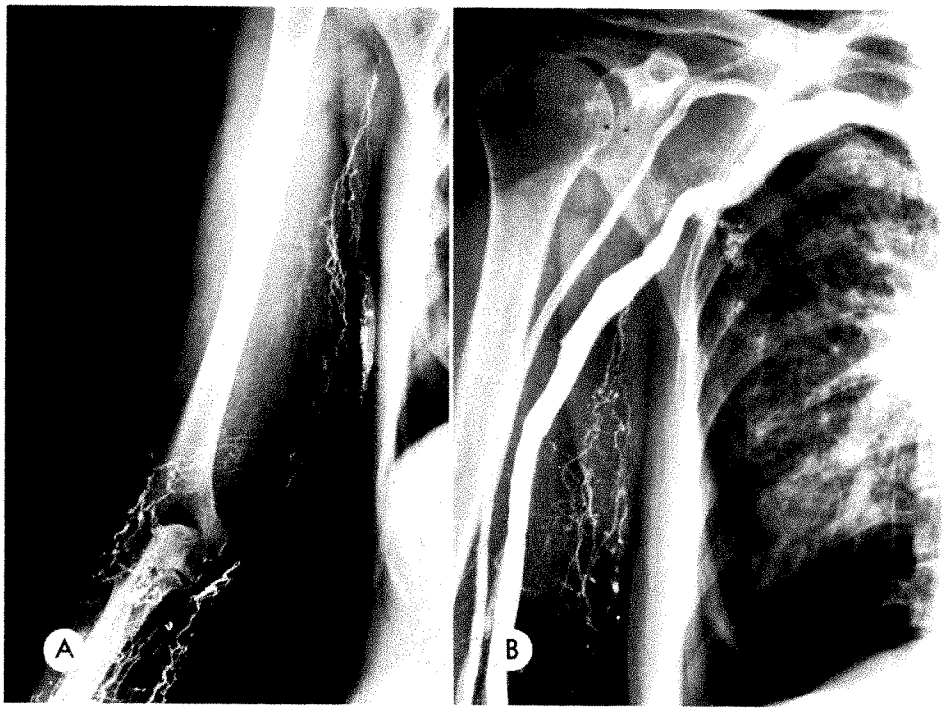


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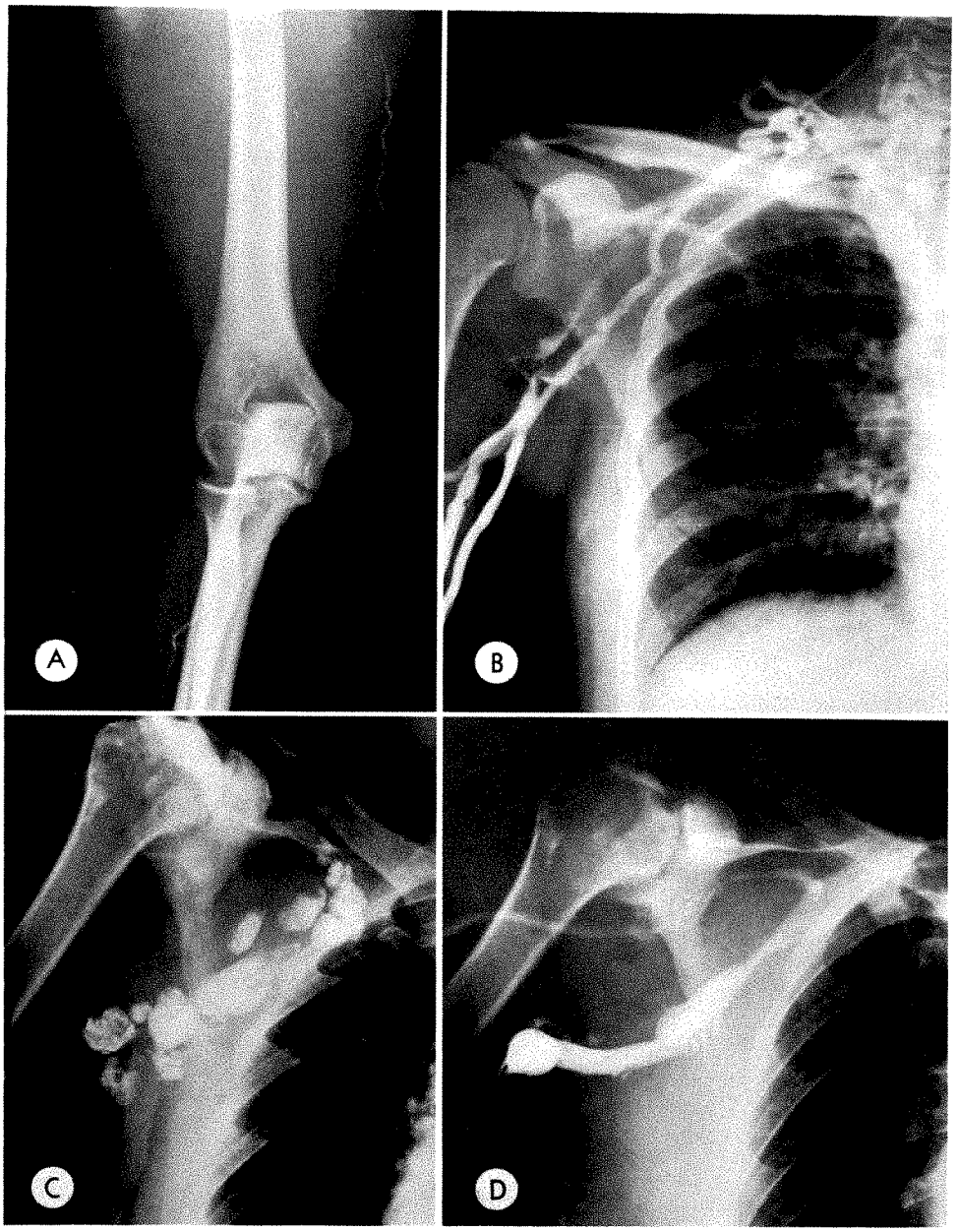


FIG. 13. *Edema of the arm due primarily to venous occlusion.* (*A*) The lymphangiogram of a patient with postmastectomy edema demonstrates a single fine lymphatic channel. (*B*) The venogram of the patient in *A* demonstrates chronic thrombophlebitis, intrinsic disease. (*C*) Lymphangiogram of another patient, studied because of edema of the arm associated with a carcinoma of the breast. The lymphangiogram reveals enlarged axillary lymph nodes. (*D*) Venogram of patient in *C* shows extrinsic compression of the axillary vein. (Schaffer and Gould, Philadelphia General Hospital.)

obstruction. The site of the obstruction was in the axilla. Multiple collateral channels are clearly seen attempting to circumvent the obstruction. The lymphatics are dilated and tortuous with associated dermal backflow. The venogram (Fig. 12*B*) was normal.

2. *Venous Occlusion.* Venous occlusion may be either intrinsic (thrombophlebitis) or extrinsic (vascular compression) in ori-

gin. The patient shown in Figure 13, *A* and *B* had a carcinoma of the breast which was treated by radical mastectomy and axillary dissection followed by radiation therapy. Postmastectomy edema gradually developed. The venogram revealed chronic intrinsic thrombophlebitis. Lymphangiograms showed also a decrease in the caliber and number of the lymphatics. It is believed by many that chronic thrombophlebitis is usually coupled with perivascular lymphangitis.[52] In an attempt to explain our findings, it is postulated that there is stasis in the lymphatics of the extremity, thereby making fewer channels available for active flow. On the other hand, perhaps the decrease in caliber is secondary to the increase in interstitial tissue pressure stasis or spasm.

Extrinsic venous disease may be due to enlarged lymph nodes, inflammatory or neoplastic in origin, compressing the veins. It may also be produced by fibrotic or inflammatory changes in the perivascular tissues secondary to surgery or radiation therapy and may eventuate in an occlusion of the vein. Figure 13, *C* and *D* shows extrinsic venous disease in a patient with a carcinoma of the breast. There was edema of the arm prior to any form of therapy. The lymph nodes compressing the vein proved to be inflammatory.

3. *Combined Lymphatic and Venous Disease.* Most frequently encountered is a combination of these two entities. In such situations the vein is involved either by intrinsic or extrinsic disease. Lymphangiograms show lymphatic obstruction with extensive collateral circulation. However, the lymphatic vessels are finer in caliber than those seen with "pure" lymphatic obstruction. There is usually an associated soft tissue suffusion of contrast material.

Obstruction of the superior vena cava and right subclavian vein by a carcinoma of the lung may also occlude lymphatic flow (Fig. 14, *A* and *B*). The increased

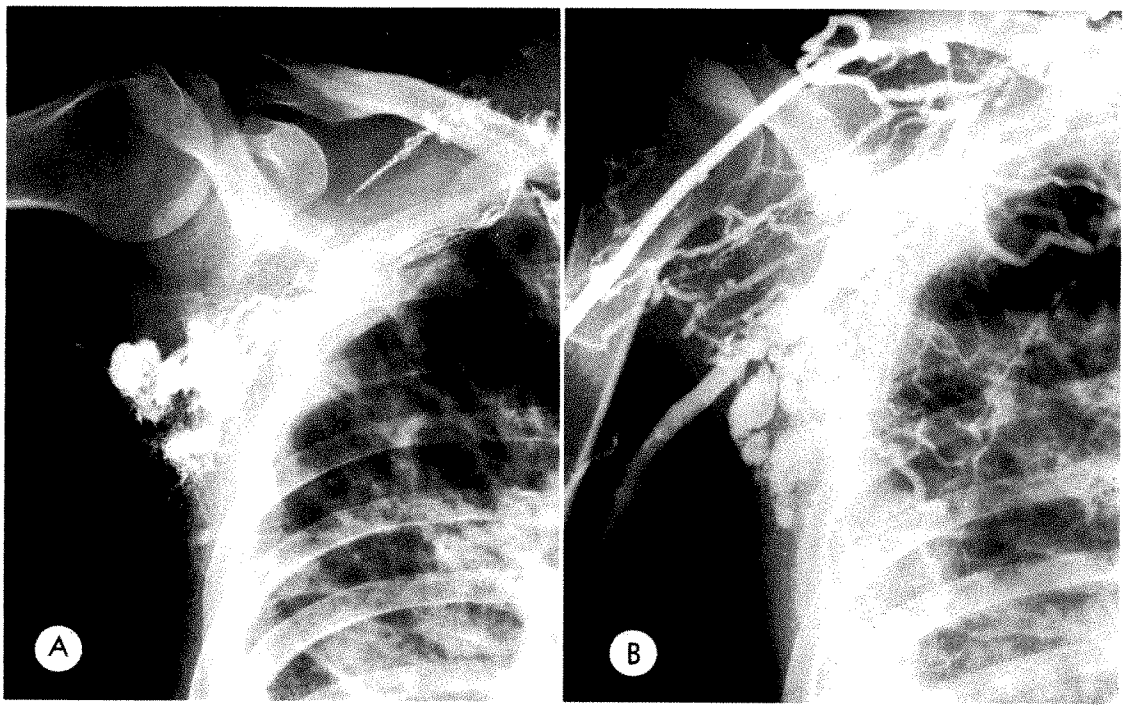


FIG. 14. *Combined lymphatic and venous disease in the production of edema.* (*A*) This patient with a carcinoma of the lung was studied because of edema of the right upper extremity. The lymphangiogram demonstrates lymphatic obstruction with associated collateral circulation in the axilla. (*B*) The venogram reveals occlusion of the subclavian vein with concomitant venous collaterals. (Schaffer and Gould, Philadelphia General Hospital.)

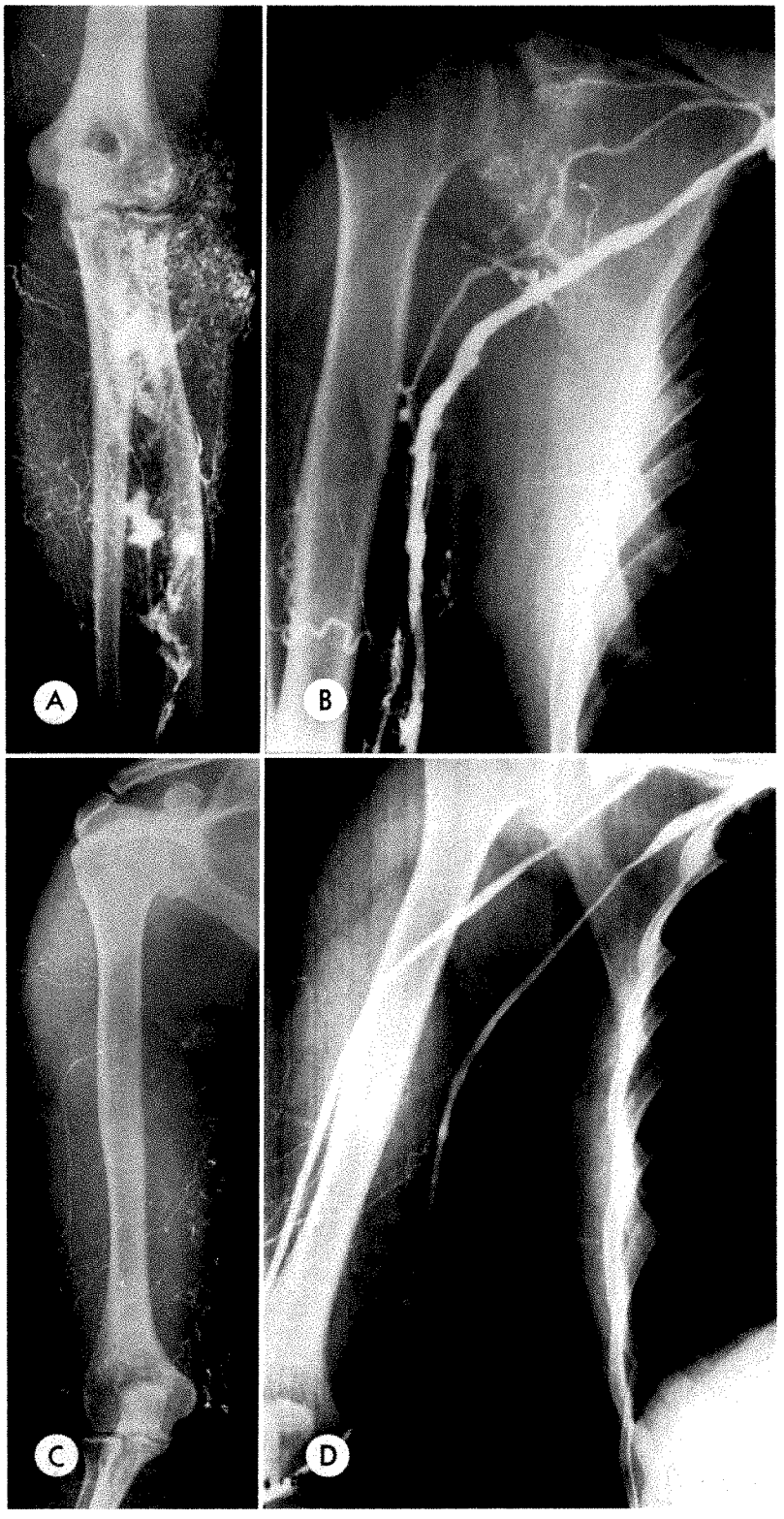


FIG. 15. *Combined lymphatic and venous disease.* (*A* and *B*) This patient with Hodgkin's disease was treated by radiation therapy following a biopsy of an axillary lymph node. Progressive edema ensued. The lymphangiogram demonstrates extensive collaterals with some suffusion of contrast into the interstitial tissue. The venogram reveals narrowing and irregularity of the axillary vein probably secondary to chronic thrombophlebitis. (*C* and *D*) Another patient with edema of the upper extremity following radical mastectomy and radiation therapy demonstrates the combination of lymphatic obstruction and extrinsic venous compression

venous pressure inhibits the normal flow of lymph into the subclavian vein at the venous angle. The lymphangiogram shows marked collateral lymphatic circulation in the axillary and supraclavicular areas and in the lateral chest wall. There are irregular supraclavicular lymph nodes demonstrated which are diagnostic of metastatic disease. Venograms demonstrate filling of accessory venous channels bypassing the occlusion.

Figure 15, *A* and *B* shows a patient with Hodgkin's disease and extensive axillary lymph node involvement. The diagnosis was established by axillary lymph node biopsy and followed by radiation therapy to the axilla. There was progressive edema of the arm. The lymphangiogram shows an extensive collateral lymphatic network; these vessels are, in general, fine in caliber and there is soft tissue suffusion of the radiopaque material. The venogram demonstrates an irregular axillary vein, suggesting thrombophlebitis.

Another patient with extrinsic venous disease in combination with lymphatic obstruction is shown in Figure 15, *C* and *D*. This patient developed edema of the arm following a radical mastectomy and radiation therapy. The axillary and cephalic veins are tapered in the axilla, suggesting extrinsic compression. The lymphangiogram reveals extensive collateral circulation through lymphatics of fine caliber and slight soft tissue suffusion of the contrast material. The lymphangiographic picture in such instances has been consistent.

SUMMARY

The study of lymphatic dynamics has been made possible by lymphangiography. Fluid dynamics of the blood vascular system are applicable to the lymphatic system even though the rate of flow is markedly decelerated.

Alternative routes of flow in certain abnormal states have been described. Obstruction to lymphatic pathways results in the establishment of collateral circulation through the most available channels in order to circumvent the interruption and maintain the integrity of the system. When such mechanisms are inadequate, there is an accumulation of relatively static fluid, resulting in edema. Other alternative avenues lie in the neurovascular sheaths and lymphatic-venous anastomoses. The intimate relationship between the lymphatics and veins must be appreciated before the problem of the edematous extremity can be understood. These are but a few of the mechanisms employed in the maintenance of lymphatic flow.

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A PHYSIOLOGIC APPROACH TO LYMPH FLOW IN LYMPHOGRAPHY*

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LYMPHOGRAPHY has enjoyed and suffered the consequences of sudden popularity. Intralymphatic injection of oily contrast media provided spectacular opacification of the lymph nodes. The enthusiasts hoped to detect subclinical lymph node metastasis. When it became evident that this was not feasible, many abandoned the procedure as useless. In reality, the most valuable information obtained by injection of oily substances concerns the lymph flow. However, maintenance of the lymph flow is the major physiologic role of the lymphatic system. We believe that a new era of clinical lymphography is about to start, wherein the emphasis on the morphology of the lymph nodes will shift to a dynamic approach to the lymphatic system, primarily toward the study of the characteristics of lymph flow.

Deviations from normal pathways of lymph flow at lymphography have been found in our experience to be a sensitive, reliable index of abnormalities of the lymphatic system. The manifestations of these abnormalities are two-fold: obstruction of usual channels and appearance of alternate pathways.

Kinmonth *et al.*,[6] who developed the present technique of lymphography, studied edema of the lower extremities. They described alternate pathways of lymph flow in edema secondary to lymphatic obstruction. Greening and Wallace[3] emphasized the evaluation of channels in the interpretation of lymphograms. Picard and Manlot[9] pointed out obstruction and deviation of channels and suggested the existence of lymphaticovenous, lymphaticoportal, and pyelolymphatic communications. Other workers have described alternate pathways of lymph flow in the abnormal lymphogram.[5,11]

To analyze lymph flow it is important to recognize that the lymphatic apparatus is composed of (A) the lymphvascular system and (B) the lymphoid tissues (lymph nodes, spleen, bone marrow, etc.).

This concept of duality is based on the following:

1. Embryologically and phylogenetically the lymphvascular system appears earlier and separate from lymphoid tissue.[10,12]

2. Anatomically, the lymphvascular system is widespread throughout the body while the lymphoid tissues are localized structures.[10]

3. Physiologically, the function of the lymphvascular system is the return of extravascular protein to the venous circulation, a function essential for life. The function of the lymphoid tissues, whatever it may be, is separate from that of the lymphvascular system.[10,12]

4. Experimentally lymphoid tissue can be destroyed by irradiation with no effect on rate of lymph flow.[1,7]

In the roentgenographic study of the lymphatic system, the information obtained depends partly on the contrast material used.[2,8]

a. Crystalloids (water soluble materials), while miscible in lymph, diffuse out rapidly from the lymphatic system. They can be used to study the lymphatic channels of the extremities, but not of the entire system.

b. Colloidal substances (oils, Thorotrast, etc.) are immiscible in lymph but are retained in the lymphvascular system and

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therefore are very useful in studying lymph flow in the vessels. Oils are also retained in the lymph nodes, but, because they are immiscible in the lymph and form globules of various size, they do not demonstrate the fine structure of the lymph node.

c. Corpuscular material (india ink, red blood cell, etc.) is also retained in the lymphatic system and is useful to study the filtering capacity of the lymphoid tissues.[12] Its flow characteristics are not known to us.

Ethiodol, the contrast medium presently in use, behaves as a colloid and is ideal for studying the lymphatic channels. The duality of the system is apparent when one studies the immediate postinjection lymphograms wherein the channels are visualized first and independently from the lymph nodes (Fig. 1, *A* and *B*).

Illustrative cases showing abnormality in lymph flow and physiologic and anatomic approach to lymphographic analysis are presented.

ILLUSTRATIVE CASES

CASE I. S.K., a 51 year old female, presented with marked postmastectomy edema of the left upper extremity. Two years previously she underwent a left radical mastectomy for infiltrating duct cell carcinoma with axillary lymph node metastases. She had received postoperative radiation therapy to the mediastinum and supraclavicular-axillary regions. Venography was essentially normal. Lymphography was performed.

The lymphogram (Fig. 2) showed that the usual lymphatic channels were completely absent. Many fine tortuous dermal collaterals were demonstrated; the tubular structures which were visualized represented extensive perivascular lymphatic opacification. On the delayed roentgenogram only a single lymph node was visualized in the cubital region. A large amount of contrast material was retained in the collaterals and perivascular lymphatics for weeks.

Comment. This case shows that following occlusion of the usual pathways, dermal collateralization occurs; apparently when the dermal collaterals are inadequate the perivascular pathways become opacified.

CASE II. A.Z., a 60 year old male, was admitted because of increasing weakness, fever, and malaise. Ten months prior to admission, the patient received radiation therapy to an enlarged right neck lymph node, which sub-

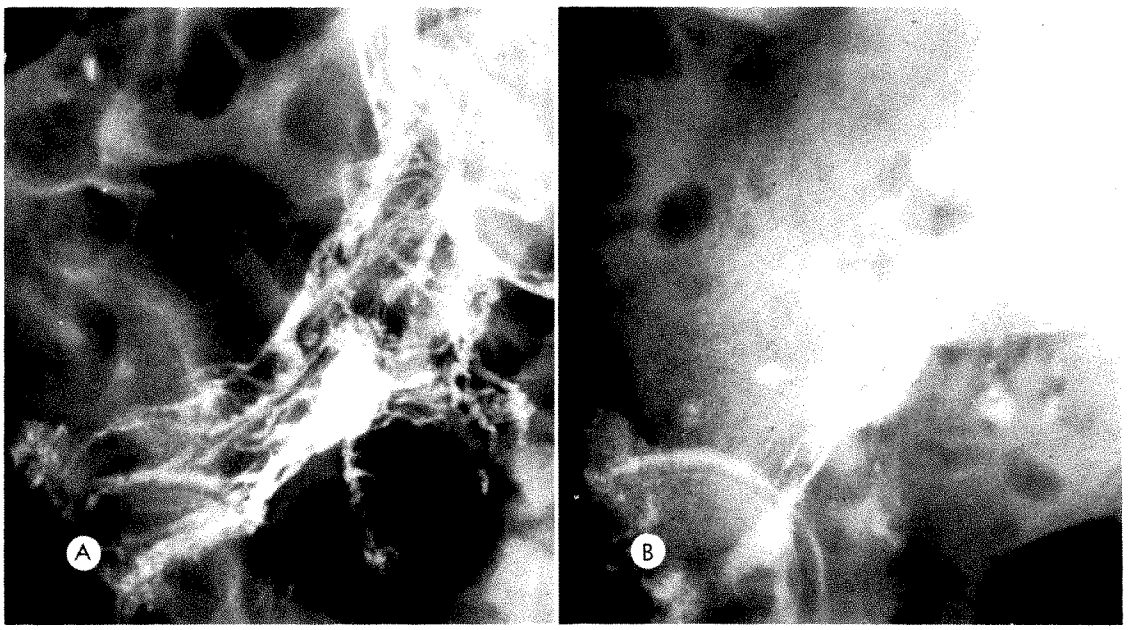


FIG. 1. (*A*) Immediate postinjection roentgenogram shows opacification of the abundant lymph channels independently from lymph nodes. (*B*) On the 24 hour roentgenogram the lymph nodes are visualized.

sequently regressed to normal size. Physical examination showed only minimal hepatosplenomegaly. Small palpable inguinal lymph nodes were present. Blood urea nitrogen was 106 mg. per cent. Retrograde pyelography revealed bilateral ureteral obstruction. Inguinal and axillary lymph node biopsies showed only lymphoid hyperplasia. The lymphogram was consistent with abdominal lymphoma obstructing the ureters. Ureteral catheters were introduced and radiation therapy was given, 2,500 r to the tumor in 3 weeks. Following irradiation, the lymph nodes became smaller, the blood urea nitrogen gradually returned to normal and the patient became asymptomatic.

Lymphograms (Fig. 3; and 4, *A* and *B*) showed prominent collateral channels extending from the inguinal region into the scrotum and across the lower pelvis. Innumerable collateral channels were noted in the iliac regions,

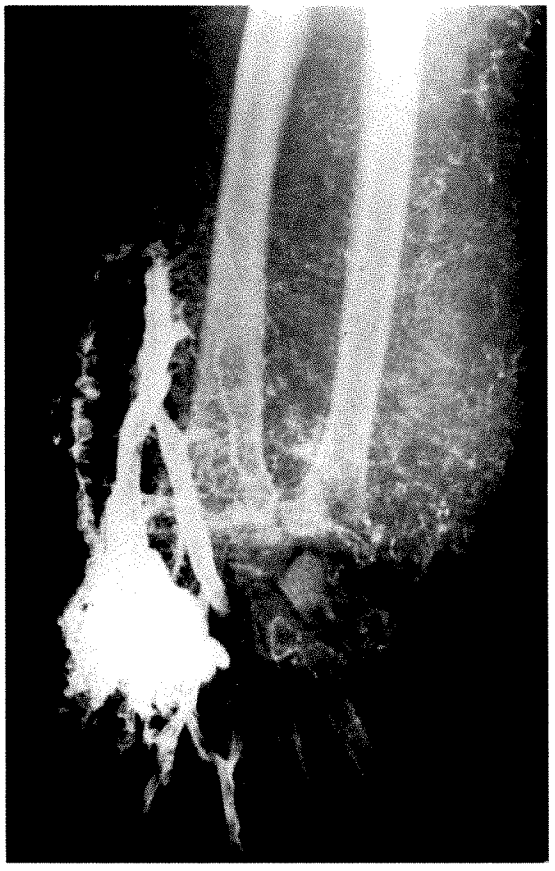


Fig. 2. Case I. Postmastectomy edema. Absence of usual lymphatic channels, with many tortuous collaterals and extensive perivascular lymphatic opacification is demonstrated.

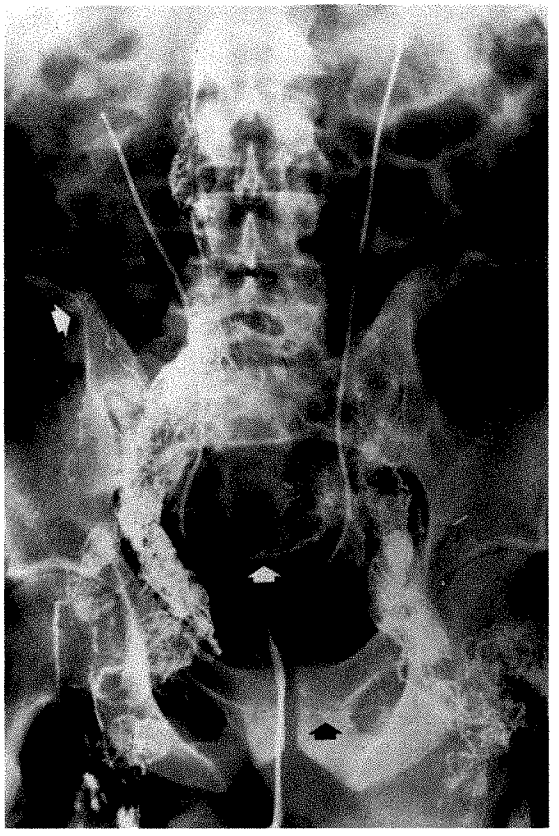


Fig. 3. Case II. Arrows point to collateral channels in the interinguinal, pelvic and right lumbar regions. Innumerable collateral channels are replacing the usual iliopelvic pathways.

while in the juxta-aortic area only a few channels were opacified. Some unusual channels in the aortic area led to the kidney region. The large lymph nodes presented a lacy lymphomatous pattern.

Comment. The marked lymphatic collateralization was due to functional obstruction which is unusual in lymphoma. It is noteworthy that along the path of collaterals no lymph nodes were seen.

Case III. G. G., a 69 year old man, presented with a several months' history of left sided abdominal pain. A left sided flank mass was suspected by palpation. An intravenous pyelogram showed some displacement of the left ureter. An abdominal aortogram was normal. Lymphography revealed extensive disturbance of the lymphatic system. At subsequent exploration, the entire retroperitoneum was found to be involved by undifferentiated carcinoma.

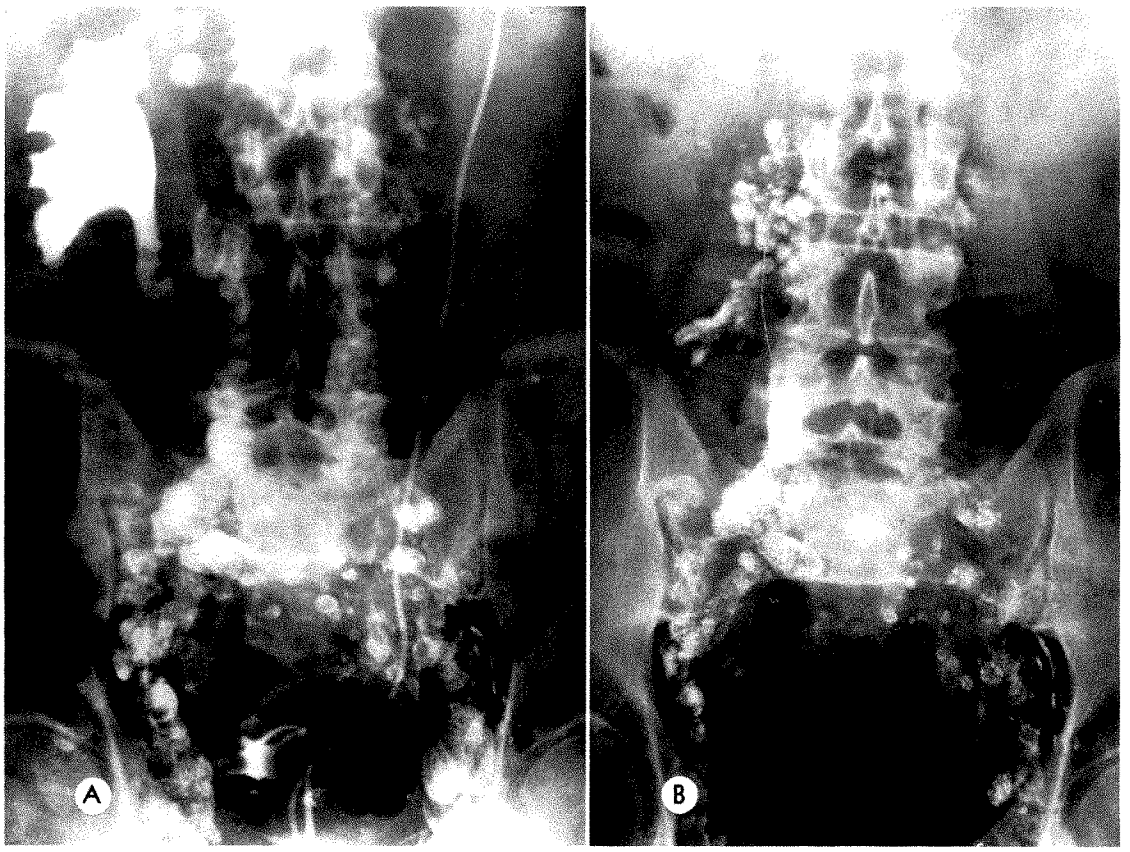


FIG. 4. Case II. (*A*) The lacy pattern of the enlarged lymph nodes is characteristic of lymphoma. The hydronephrosis is secondary to extrinsic pressure. Note the absence of lymph nodes along the path of collaterals in the lumbar and pelvic regions. (*B*) Following irradiation, there is marked decrease in size of the lymph nodes.

The lymphogram (Fig. 5) showed that the usual lymphatic pathways in the iliopelvic aortic regions were absent. Only irregular small collaterals were opacified and were displaced anteriorly and laterally. Larger channels devoid of lymph nodes extended to the juxta-aortic region. The only lymph nodes visualized were in the inguinal area.

Comment. This case shows that when the normal pathway of lymph flow is obstructed alternate pathways which do not traverse lymph nodes become opacified.

CASE IV. A.S., a 75 year old male, presented with an increasing mass in the right groin of 1 year's duration. Marked swelling of the right lower extremity was present; the left lower extremity was also edematous. Biopsy of a right inguinal lymph node showed Hodgkin's sarcoma. Lymphography was performed. The patient expired 3 months later and autopsy revealed Hodgkin's disease involving the iliopelvic, aortic, and inguinal lymph nodes, right kidney, liver and right testis. There was bilateral occlusion of the iliac veins.

The lymphogram (Fig. 6) showed a complete obstruction to the lymph flow at the ilioinguinal junction on the right side. Collateral pathways crossed over from the right to the left inguinal region. The delayed roentgenograms showed no lymph nodes on the right side above the inguinal region, while the lymph nodes of both inguinal and the left iliac regions had large filling defects. Their foamy appearance was consistent with Hodgkin's disease.

Comment. In this case autopsy confirmed extensive involvement by Hodgkin's disease of the iliac regions bilaterally. It is apparent that the interinguinal collaterals were due to this obstruction.

Case v. H.H., an 83 year old male, presented with back pain and edema of the lower extremities. Three years previously prostatectomy was performed for "benign prostatic hyperplasia." A roentgenographic skeletal survey subsequently showed widespread osteoblastic metastases. Intravenous pyelography revealed left hydronephrosis. Lymphography was performed.

The lymphogram (Fig. 7) showed normal appearing channels bilaterally in the inguinal regions. In the lower external iliac area the lymphatic channels were markedly dilated, and many collateral pathways were opacified. A large lymphatic channel led to a left lateral sacral lymph node. A complete block of lymph flow was present at the common iliac level. On the postinjection roentgenograms, pools of contrast material were seen in the lymph nodes, suggesting dilated sinusoids in enlarged lymph nodes. The delayed roentgenograms showed distortion of anatomic distribution of the enlarged irregularly filled lymph nodes, again revealing pools of contrast material within.

Comment. This case demonstrates the effect of malignant extension blocking the lymph flow. This manifested itself in dilatation of the usual channels, collateralization and distortion of intranodal lymphatic pathways (dilated sinusoids).

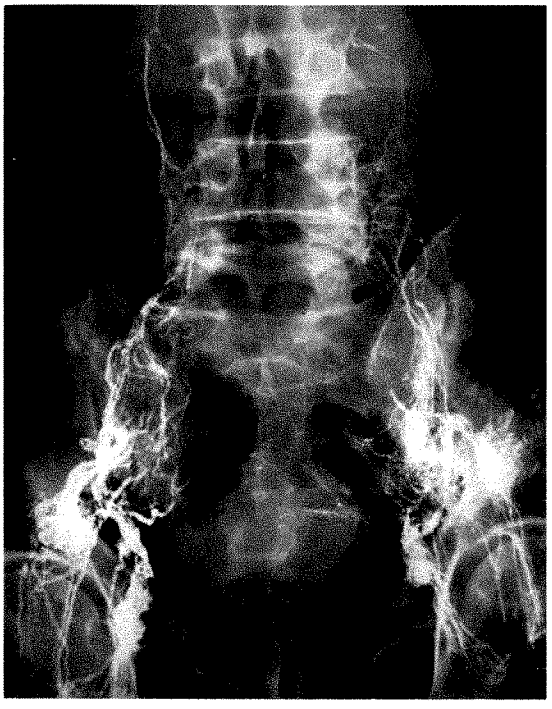


Fig. 5. Case iii. The usual iliopelvic-aortic lymphatic pathways are absent; only irregular collaterals are opacified. On delayed studies, no lymph nodes were visualized along these collaterals.

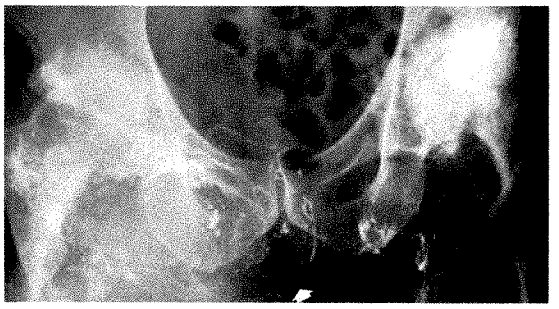


Fig. 6. Case iv. Hodgkin's disease. Arrow points to an interinguinal collateral which is indicative of functional obstruction in the system.

Case vi. V.S., a 55 year old man, was readmitted to the hospital with increasing dyspnea of 1 month's duration. He had been hospitalized 1 year previously when a histologic diagnosis of lymphoma of the retroperitoneal space with periureteral fibrosis was made. He received radiation therapy to the entire abdomen, 3,000 r tumor dose in 4 weeks.

A chest roentgenogram on present admission showed a huge right pleural effusion, which proved to be chylous. Lymphography was performed. The patient was treated with intra-

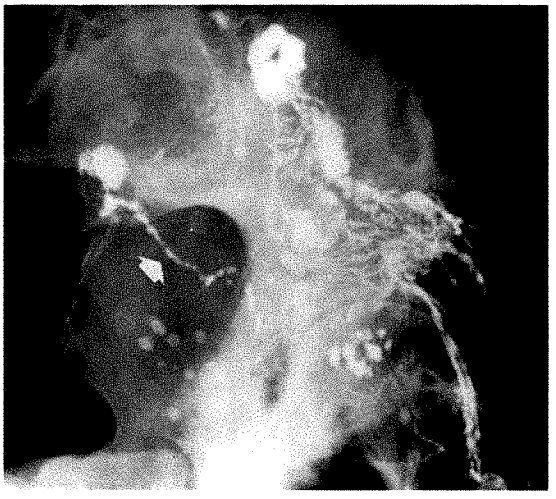


Fig. 7. Case v. Arrow points to a prominent channel leading to a lateral sacral lymph node. Note the dilated sinusoids in the lymph nodes due to a complete obstruction at the common iliac level.

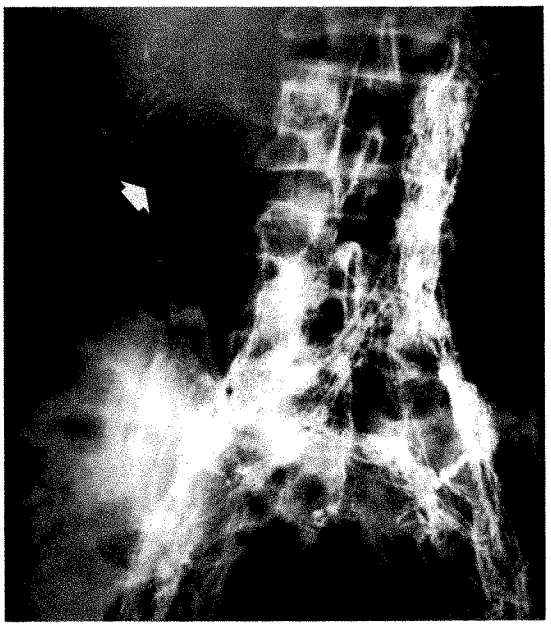


FIG. 8. Case VI. Superabundance of collaterals in the iliac regions is seen. The right juxta-aortic channels are largely absent, but arrow points to unusual collaterals extending to right lumbar region.

pleural nitrogen mustard with subsequent improvement.

Lymphograms (Fig. 8; 9, *A* and *B*; and 10) showed a superabundance of collateral channels in the iliac regions with marked crossing over to the left side. The right juxta-aortic channels were largely absent, but unusual collaterals extended to the right as far as 10 cm. Many fine caliber channels were opacified in the left juxta-aortic region. The thoracic duct was well visualized and was normal. There was marked diminution in size and number of the lymph nodes in the iliopelvic and lower aortic regions bilaterally; some abnormal lymph nodes were visualized in the left upper juxta-aortic area.

Comment. This case shows that the chylothorax was not due to obstruction of the thoracic duct, but to a block in the juxta-aortic area with collateral paths leading to pleural space. The previous radiation therapy to the entire abdomen caused marked diminution of the lymphoid tissues (lymph nodes) but no apparent change in the lymph flow.

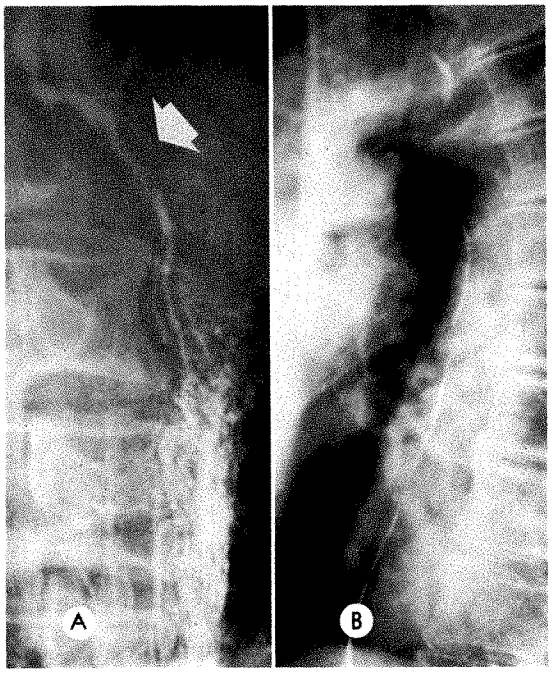


FIG. 9. Case VI. (*A*) The cysterna chyli and the thoracic duct have their origin entirely from the left juxta-aortic channels (arrow). (*B*) In spite of a right chylothorax, the thoracic duct is normal.



FIG. 10. Case VI. In spite of abundant lymphatic channels in the iliopelvic region (Fig. 8), there is a lack of lymph nodes along the path of these collaterals.

Case vii. B.L., a 50 year old male, was admitted because of an increasing right testicular mass of 3 months' duration. Subsequently, he underwent a right orchidectomy. The pathologic diagnosis was embryonal cell carcinoma without cord involvement. Lymphography was performed. The patient received radiation and chemotherapy and expired 1 month later.

Lymphograms (Fig. 11 and 12) demonstrated several unusual lymphatic channels in the iliopelvic region. At the level of the third lumbar vertebra, complete cut-off of the lymph flow on the right side was noted, and via collateral pathways the entire liver was opacified in a diffuse reticular pattern. On the delayed roentgenograms no lymph nodes could be identified in the juxta-aortic region and irregularly filled, disorganized lymph node groups were seen in the iliopelvic regions.

Comment. The apparent complete obstruction of the lymph channels in the juxta-aortic region diverted the lymph flow toward the liver. It is uncertain whether the contrast material was in intrahepatic lymphatics or in portal vessels. This patient

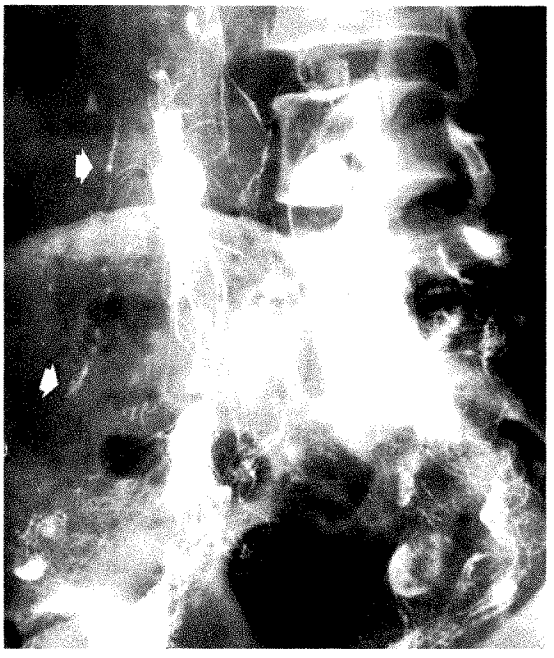


Fig. 11. Case vii. Unusual lymphatic channels (arrows) are seen in the iliopelvic-aortic region. There is a complete block of lymphatic channels at the juxta-aortic region, and flow is diverted to the liver (Fig. 12).

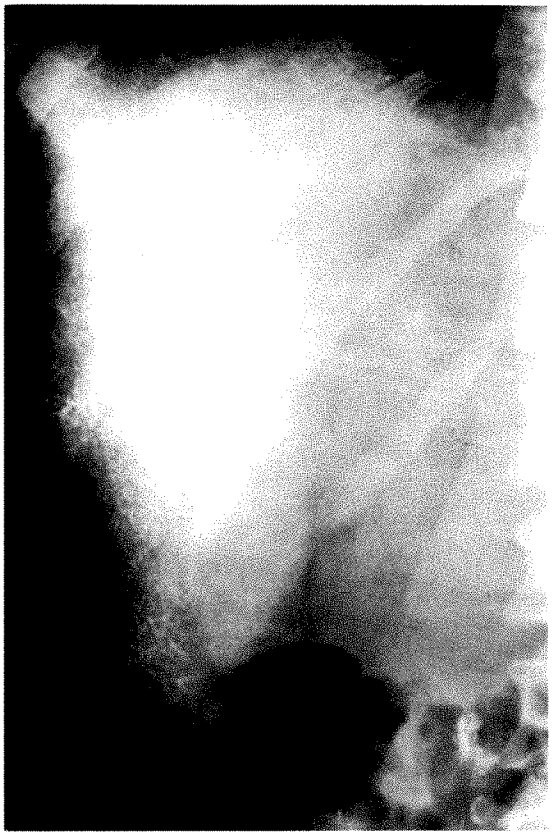


Fig. 12. Case vii. Diffuse opacification of the liver is seen.

was followed for several days during which time the material remained in the liver. It is therefore assumed that intrahepatic lymphatic channels had been opacified.

Case viii. I.M., a 34 year old female, was admitted for radiation therapy of a Stage ii carcinoma of the uterine cervix. She received external radiation and intrauterine radium insertion. Five months later local recurrence was noted. Prior to radical hysterectomy and pelvic lymphadenectomy, lymphography was performed. The patient was operated on and had an uneventful recovery. Three months later lymphography was repeated. The patient was and is at present asymptomatic.

A preoperative lymphogram (Fig. 13*A*) showed normal channels and lymph nodes in the entire iliopelvic aortic lymphatic system. A postoperative lymphogram showed that all the external and lower common iliac lymph nodes and the hypogastric lymph nodes had been removed. Repeat lymphography 3 months fol-

lowing operation showed a lymphocyst, 3 cm. in diameter, in the left external iliac region. On delayed roentgenograms many collaterals were seen emerging from the lymphocyst (Fig. 13*B*).

Comment. This case shows an alternative for lymph flow following surgical transection of major lymphatic trunks—lymphocyst formation. In this case collaterals emerging from the lymphocyst maintained lymph outflow and the patient remained asymptomatic.

DISCUSSION

In order to appreciate alterations in lymph flow, it is essential to understand the normal roentgen anatomy of the lymphatic channels and lymph nodes. This normal roentgen anatomy for the iliopelvic aortic regions has been previously described.[4]

The cases we have presented demonstrated the following alteration in lymph flow:

a. dermal collaterals
b. perivascular pathways
c. alternate channels not associated with lymph nodes replacing the normal iliopelvic-aortic channels
d. diminution in size of lymph nodes after irradiation, with maintenance of lymph flow
e. dilatation of intranodal lymph pathways secondary to obstruction
f. chylothorax in the presence of a normal thoracic duct, but with extensive collateralization in the iliopelvic-aortic regions
g. diffuse opacification of the liver in the presence of obstruction of the upper juxta-aortic channels
h. lymphocyst formation following surgery

On lymphograms the appearance of unusual pathways shows that there is a significant disturbance of the lymph flow.

Obstruction and collateralization often occur in a complementary fashion; obstruction can be differentiated from incomplete filling by the presence of collaterals. It is important to realize that these alternate

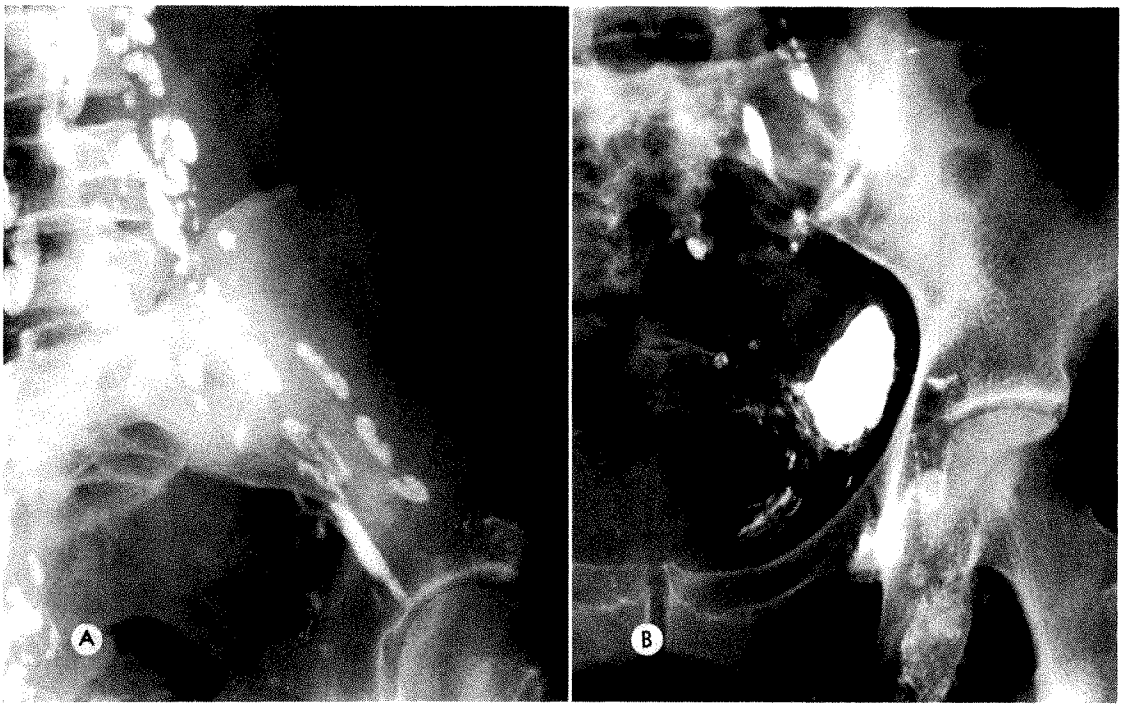


FIG. 13. Case VIII. (*A*) Preoperative lymphogram demonstrates normal lymph nodes in the iliopelvic region. (*B*) Lymphocyst is seen after pelvic lymphadenectomy. Collaterals emerge from the lymphocyst.

pathways are normally existing and probably functioning channels. They do not opacify with the present method of lymphography when the system operates at normal capacity.

It is apparent that in lymphographic analysis a systematic approach should be based on the following concepts:

1. The realization that the information obtained depends on the properties of the contrast medium.
2. Knowledge of the normal roentgen anatomy with an understanding that only a fraction of the vast lymphvascular system is usually opacified.
3. The appreciation of the duality of the lymphatic apparatus which consists of the lymphvascular system and the lymphoid tissues. In clinical lymphography, evaluation of lymph channels and lymph flow is essential.

SUMMARY

Eight cases showing alteration of lymph flow in lymphograms were analyzed. It was concluded that the information obtained depends on (1) properties of the contrast media, (2) understanding of the normal roentgen anatomy of the lymphvascular system, and (3) appreciation of the duality of the lymphatic apparatus which consists of the lymphvascular system and the lymphoid tissues. In clinical lymphography, evaluation of lymph channels and lymph flow is essential.

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LYMPHANGIOGRAPHY—A SURVEY OF ITS CURRENT STATUS*

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THE current literature reflects an increasing interest in lymphangiography. Since opinions vary widely as to the value and place of lymphangiography in patient management, it was decided to send a questionnaire on this procedure to various investigators in order to assemble opinion and experience in a significant group of patients. The results of this survey representing experience with more than 4,000 patients are presented below. It is felt that these results are of value in offering a guide to those whose experience with lymphangiography may still be somewhat limited, and to acquaint others with the present consensus regarding this procedure.

METHOD

One hundred and forty-one questionnaires were sent to investigators in the United States, England, France and Sweden. These men had written about or were believed to have had experience with lymphangiography. The questions related to the present technique, the clinical indications, value, and expected accuracy in different diseases, and the complications of the procedure. As with most questionnaires, the answers reflect general impressions rather than detailed case analysis. This must be borne in mind in evaluating the results.

From 80 questionnaires returned, only 29 reflected experience with sufficient cases to be judged valid for this report. While some investigators reported on many hundreds of cases, all answers reporting on experience with more than 25 cases were accepted. The results include experience with 4,260 patients, 184 being under 18 years of age.

The questionnaire was divided into three parts: (1) technical aspects of the procedure; (2) interpretation of the study; and (3) complications of the procedure.

RESULTS OF THE QUESTIONS DEALING WITH TECHNIQUE

All investigators who responded to the questionnaire are using the method described by Kinmonth *et al.*[8] with various technical modifications. Except for the Swedish investigators who employ water soluble contrast solutions, the majority use ethiodol or chlorophylated ethiodol, the total dose being 15 to 20 cc. for bilateral leg injections in adults. In children no more than 15 cc. per study was used. A few European centers prefer lipiodol.

Many vital dyes are injected intradermally to stain the lymphatics for identification purposes. The most commonly employed was Evans Blue. Others used were Patent Blue, Methylene Blue, Sky Blue, and Brilliant Blue-F.D.C.#1. The maximum duration of skin staining was 4 months. While some investigators feel that Brilliant Blue-F.D.C.#1 is the best dye, this has not been approved for use, as of this writing, by the Food and Drug Administration.

Most investigators use an automatic injector taking from 15 minutes to 4 hours for the injection. The ideal speed of injection appears to be 45 minutes to 90 minutes. Others find the manual screw or weight-injector satisfactory. After evaluation of some of the automatic injectors, we feel that the most desirable are those designed with a safety pressure gauge behind a syringe, in such a manner that the syringe can be connected directly to the plastic injector tube, thus avoiding certain sterilization problems that exist with a type of injector that has a fixed metal reservoir

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interposed between the syringe and tube (Fig. 1).

While the vast majority of injections are in the dorsum of the feet, about half of the investigators had occasional cases in which the upper extremity was studied. Only 2 investigators reported using the procedure in head and neck cases.

Premedication was frequently employed by about one half of the investigators. Although the procedure itself is not particularly uncomfortable for the patient, the premedication is used to allay apprehension.

About half of those reporting perform lymphangiography on an outpatient basis. While individual studies vary, lymphangiograms are routinely taken immediately after the end of injection, and at 24 hours. In very few instances roentgenograms were also taken at 48 hours.

Fourteen of the twenty-nine investigators have obtained a second lymphangiogram of the same area. Half of those answering this question felt that the repeat study is somewhat more difficult than the original. This is because of the disruption of the local lymphatics at the site of injection by the first procedure. Thirteen of this group felt that repeat studies reliably show progression or regression of disease in the lymph nodes.[9] No one found evidence of damage to lymph vessels or lymph nodes which was attributed to the previous lymphangiography. This emphasizes the experience previously reported by our group,[10] that the oily contrast material does not cause permanent damage in the lymphatic system. This conclusion was based on observations over a period of over 2 years.

RESULTS OF THE QUESTIONS DEALING WITH ACCURACY OF INTERPRETATION

Basic to the interpretation of the lymphangiogram is the problem of how many lymph nodes in an area are demonstrated by the procedure. This has been studied in surgical and autopsy correlation,[3,5] with varying opinions expressed. The question

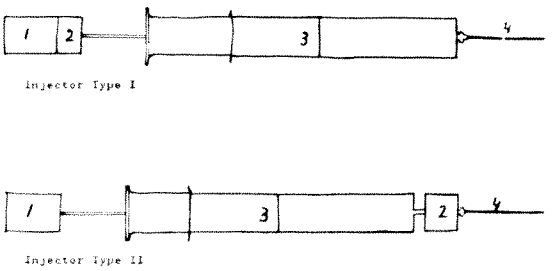


Fig. 1. Schematic drawing of the two types of automatic injectors. In Type I a direct connection between the syringe and tubing is present. In Type II a pressure regulation mechanism, which cannot be sterilized, is interposed between syringe and tubing. (1) Driving motor, (2) transducer of pressure regulator, (3) syringe, and (4) tubing.

was therefore asked "What is the number of lymph nodes you believe can be opacified in the following area?" The following answers were received:

	Few	Most	All	No answer
Pelvis	1	24	2	2
Retroperitoneal	1	21	4	3
Thorax (mediastinum)	24	0	0	5
Axilla	5	12	0	12

From this table it is apparent that most investigators feel that they opacify most but not all of the lymph nodes in the pelvis, retroperitoneal area and axilla. Only very few lymph nodes in the mediastinum are visualized.

It has been demonstrated that there are certain characteristic patterns of ethiodol deposition in the lymph nodes in lymphoma, carcinoma, and in inflammatory and other benign diseases.[10,11] However, while in the majority of cases, characteristic patterns make roentgen diagnosis of the disease type possible, there are not infrequent occasions in which one pattern may simulate another.[10] We were interested in the experience of others in differentiating not only the major classifications of disease, but the subgroups within a classification, *e.g.*, Hodgkin's disease versus lymphosarcoma, a differential diagnosis that usually can be made histologically. The question was

therefore asked "Can lymphangiography usually distinguish between the following: (a) various benign inflammatory diseases; (b) types of lymphedema; (c) types of lymphoma; and (d) types of carcinoma?" The following answers were obtained:

	Yes	No	No answer
(a) Various benign inflammatory diseases	2	17	10
(b) Types of lymphedema	20	3	6
(c) Types of lymphoma	9	16	4
(d) Types of carcinoma	2	25	2

From the answers it is apparent that most investigators have not found it possible to subdivide inflammatory diseases, lymphomas, and carcinomas into finer classifications. This also is our experience.

In lymphedema specific patterns of lymphatic anatomy have been found which clarify the underlying pathology. This important contribution was confirmed by almost all investigators. For further information, the reader is referred to the cited references where this problem is covered in great detail.[6,7,11]

There is a variation of the accuracy in the interpretation of the lymphangiograms as published by different investigators. In answer to the question "What is your estimated accuracy in the interpretation of the lymphangiograms in the following diseases?" these results were obtained:

	Poor	Fair	Good	No answer
Benign disease (inflammatory, vascular)	2	9	4	14
Lymphedema	1	6	11	11
Lymphoma	0	2	22	5
Carcinoma-early (no clinical metastases known)	16	7	1	15
Carcinoma-advanced	0	8	14	7

(Poor—less than 60%; fair—60–80%; good—over 80% accuracy.)

The answers indicate that most investigators believe that the diagnostic accuracy of lymphangiography is good in lymphedema and lymphoma. From the questionnaire it is important to note the consensus that the procedure is fairly reliable in advanced carcinoma, where clinical evidence of metastasis is already present. However, there is poor correlation in early metastatic disease.

The answer to the question "What is the smallest area of metastatic disease that you can reliably detect?" indicated that with one exception, all answering (22) felt that a focus of at least 5–10 mm. in diameter was necessary before reliable roentgenologic detection was possible. This accounts, in large part, for the poor accuracy in the early detection of carcinomatous lymph node metastasis.

The investigators were asked "What are your conclusions when seeing a negative lymphangiogram?" Sixteen out of twenty-four believed that a negative lymphangiogram in lymphoma indicates that the visualized lymph nodes are free of disease. In contrast to this, 25 out of 26 felt that a negative lymphangiogram in carcinoma did not rule out metastatic involvement of the visualized lymph nodes. This of course emphasizes the limitations of the procedure due to the inability to detect metastatic foci less than 5 mm. in diameter.

A question was then asked regarding the possibility of a lymphangiogram to demonstrate: (1) unsuspected metastatic lymph nodes; (2) clinically suspected lymph nodes; and (3) the extent of lymph node involvement in both lymphoma and carcinoma. The following answers were received:

Lymphoma

Does a lymphangiogram reveal?	Seldom	Occasionally	Often	No answer
Clinically unsuspected node involvement	5	6	14	4
Clinically suspected node involvement	0	3	23	3
The total extent of metastatic involvement of the visualized area	3	3	13	10

Carcinoma

Does a lymphangiogram reveal?	Seldom	Occasionally	Often	No answer
Clinically unsuspected node involvement	8	14	2	5
Clinically suspected node involvement	1	9	11	8
The total extent of metastatic involvement of the visualized area	11	6	4	8

From these answers, it appears that in lymphoma the majority of the investigators feel the procedure is reliable in detecting clinically unsuspected lymph nodes, confirming clinically suspected lymph nodes, and in demonstrating extent of involvement. In carcinoma, only occasionally are clinically unsuspected lymph nodes demonstrated. Where one clinically suspects metastasis, the results, while still not as good as in lymphoma, are improved. In this group, metastatic foci are often seen in the opinions of 11 investigators, and occasionally in the opinion of 9. There is divided opinion regarding the ability of this procedure to demonstrate the total extent of disease in carcinoma.

In addition to the role of lymphangiography in diagnosis, considerable attention has been given to its value in patient management. The following two tabulations are guidelines to present thought in this respect.

(a) What is, in your opinion, the value of lymphangiography in determining the type of therapy that should be used in?

The answers received are shown in the following table:

(b) Does a lymphangiogram reveal?

	Seldom	Occasionally	Often	No answer
Lymphoma				
Progression or regression of the disease	1	3	19	6
Response to radiation or chemotherapy	1	0	21	7
Carcinoma				
Progression or regression of the disease	4	11	5	9
Response to radiation or chemotherapy	6	6	5	12

Interpreting these tables, it appears that the procedure is of great help in treatment planning in lymphoma and in following progress of such treatment. In carcinoma, when positive lymph nodes are present, it is helpful in planning therapy and following progress. A negative lymphangiogram should not influence the plan of therapy.

RESULTS OF THE QUESTIONS DEALING WITH COMPLICATIONS OF THE PROCEDURE

Many questions were asked regarding complications. Since the answers were so varied, we abstracted the essential information for presentation.

Only 1 death was reported to us in 4,260 procedures. This death will subsequently be discussed in detail.

Wound infection and delayed healing occurred in 1–5 per cent of patients. This incidence emphasizes the need for strict surgical aseptic technique. Patients with

	Not important	Of some value	Should be strongly taken into account in planning treatment	No answer
Benign inflammatory disease	16	5	0	18
Lymphedema	4	11	5	9
Lymphoma	0	7	20	2
Carcinoma	3	15	6	5

edematous swollen extremities, or with impaired circulation, are more susceptible to wound infection.

Occasionally, pain is experienced during injection. This is related, as a rule, to too rapid injection of the contrast medium or to the presence of lymphatic obstruction. It usually disappears in 2 hours. In no case was pain present longer than 48 hours.

Between 5 per cent and 10 per cent of patients will have low grade fever (up to 101° F.) for periods extending to 48 hours. This fever usually disappears without treatment. Since fever does not occur with water soluble media, it may well be due to a foreign body reaction to the injected oil.

Where blockage of lymphatics was present, extravasation of the opaque medium into the adjacent soft tissues was frequent. This also occurred with too rapid injection of the contrast agent.

The presence of oil droplets in the lungs following injection has been of some concern to many working with this procedure.[1] While our studies[10] demonstrated histologic evidence of oil droplets in the lungs in every case studied in the immediate postinjection period, pulmonary oil embolization was not reported by many of those answering this questionnaire. This may be because of lack of clinical symptoms. We and others[1,4] believe, however, that oil embolization can be demonstrated in the chest roentgenogram in at least 10 per cent of patients where this study is made within 24 hours of injection. This fine granular pattern seen in the chest study usually disappears rapidly.

This pattern is in distinction to true pulmonary embolization reported in 16 cases in this series. Here, one has the classic sequence associated with pulmonary embolism from other causes.[1,4,10]

Severe bronchopneumonia developed in 3 children suffering from lymphoma within 48 hours after lymphangiography.

Because of the very interesting complications of severe pulmonary edema in 2 young men having had previous pulmonary irradiation, their histories are briefly cited.

The first patient was a 33 year old male who had lymphosarcoma with parenchymal spread to both lungs. Approximately 3 weeks prior to the study, he received irradiation to both lungs amounting to a dose of 1,700 rads. Five hours after completion of the injection of 10 cc. of ethiodol, he complained of severe shortness of breath. In 1 hour he became cyanotic, agitated and developed shock and coma. Despite intensive therapy, he died 2 hours later. A chest roentgenogram taken 1 hour before death showed bilateral pulmonary edema, but no evidence of oil embolization.

The second patient was a young man with similar history of lymphosarcoma and pulmonary parenchymal infiltration. A dose of 1,700 rads was given to both lungs several weeks prior to the lymphangiography. After the injection, he developed clinical symptoms of severe pulmonary edema similar to those in the first patient. However, in view of the experience with the first patient, intensive therapy was immediately instituted, and after 12 hours the patient recovered.

Since these are the only 2 cases of pulmonary edema reported, it is likely that the lung irradiation was directly related to the development of this complication. The exact mechanism is not known, but it has been shown that the reticuloendothelial system is responsible for removal of the oil reaching the lung,[10] and it is possible that the impaired reticuloendothelial system of the lung prevents normal oil removal and is related to pulmonary edema.

DISCUSSION AND CONCLUSIONS

From the answers to the questionnaire, it appears that the technique described by Kinmonth *et al.*[8] with various modifications is the standard method. The principal application is in the study of the pelvic and retroperitoneal lymph nodes. However, lymphangiography is also used in the study of the axillary lymph nodes and in determining the status of the lymphatic vessels. Oily contrast media are employed by most investigators, since these allow better and more complete evaluation.

From the various questions dealing with accuracy of interpretation, certain general

principles become immediately evident. Few radiologists find much application for the procedure in inflammatory diseases. It has proven of great value in determining the underlying pathology in lymphedema. In lymphoma, it has been found to be an accurate diagnostic method. It reflects the true status of the visualized lymph nodes, and is helpful in both treatment planning and in following the course of the disease.

In carcinoma, its use has been limited by our inability to detect metastatic foci less than 5 mm. in diameter, or lymph nodes completely replaced with tumor.[2] Only occasionally will a lymphangiogram demonstrate a clinically unsuspected metastatic focus. Thus, while a positive study may be helpful, a negative study is of little value for either diagnosis or treatment planning. Many surgeons find the technique useful in outlining lymph nodes in conjunction with radical surgical procedures.

Only few serious complications were encountered. These are pulmonary embolism, encountered in 16 patients, and pulmonary edema, seen in 2 patients with a history of lung irradiation in the recent pre-injection period. Minor complications were occasional wound infection, delayed healing, fever and pain.

Lymphangiography is still in the early phase of its development. It has revealed much basic information of help to the clinician, as well as to the physiologist and anatomist. New dyes and techniques may change significantly the above opinions. We would like to again stress the general nature of these questionnaire answers and encourage further application of the technique in appropriate patients.

SUMMARY

The results of a questionnaire regarding the technique, accuracy of interpretation, and complications of lymphangiography in over 4,000 patients have been presented. These results offer a guide to current thought regarding the place of lymphangiography in disease management.

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The authors wish to thank the many investigators who took the time and effort to answer the detailed questionnaire upon which this report is based. We are grateful also to Drs. Robert S. Sherman and George Schwarz of Memorial Hospital, New York City, for permission to use the 2 cited cases.

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AN ANALYSIS OF SOME ANATOMIC-ROENTGENOLOGIC ASPECTS OF THE BRACHIOCEPHALIC VESSELS*

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DURING the past few years there has been an increased interest in the study of the brachiocephalic vessels. This was stimulated mainly because of the necessity to perform total brachiocephalic arteriography in the study of patients with cerebrovascular insufficiency before an accurate diagnosis can be made and definitive therapy undertaken. The radiologist is now asked with increasing frequency for an evaluation of these vessels. A clear knowledge of the anatomy of the aortic arch and its branches, the incidence and types of common anomalies of the arch, variations in vessel size, and the effect of age on these vessels is imperative for the proper performance of the study and the evaluation of each case.

Although much work has been done on the above mentioned aspects using anatomic and autopsy specimens, little has been written using roentgenologic material. An analysis of some anatomic-roentgenologic findings of the first 100 consecutive total brachiocephalic arteriographies performed over the past 2 years at the Montefiore Hospital forms the basis of this report.

MATERIAL AND METHODS

During this period, over 150 arch aortograms have been obtained with complete visualization of all of the brachiocephalic vessels. An analysis of the first 100 cases in the series was made to evaluate the incidence and types of common anomalies of the branches of the aortic arch, the effect of age and blood pressure on these branches, and a comparison of vertebral artery size. In a few instances, a case was excluded from the series because of severe occlusive disease which would affect the anatomic evaluation of some of the vessels. When this occurred, the next consecutive case in the series was included to bring the total to 100. Seven cases were excluded for this reason.

The studies were done using the Seldinger technique of percutaneous arteriography.[16] The catheter was passed into the ascending aorta and a pressure injection of 45–50 cc. of 75 per cent hypaque (90 per cent hypaque in the later cases) was made. Rapid serial roentgenography with the Schönander apparatus was utilized. Two injections were generally performed. One series of roentgenograms was obtained of the aortic arch and its branches with the patient in the 45 degree right posterior oblique position. The second series of roentgenograms was taken over the skull with biplane filming in a Towne and lateral projection to include as much of the neck as possible.

The transfemoral approach was employed in 81 of the first 100 cases and the transbrachial approach in 19 (12 on the left and 7 on the right). In the transfemoral approach, a Kifa-Ödman yellow catheter was used in the early cases until the gray Kifa-Ödman catheter became available. In the transbrachial approach, a PE 205 or PE 240 polyethylene catheter was used. Generally, this method was employed only when there was an indication of significant peripheral vascular disease in the legs or the catheter could not be advanced through tortuous iliac vessels. At present, the transaxillary approach is employed instead of

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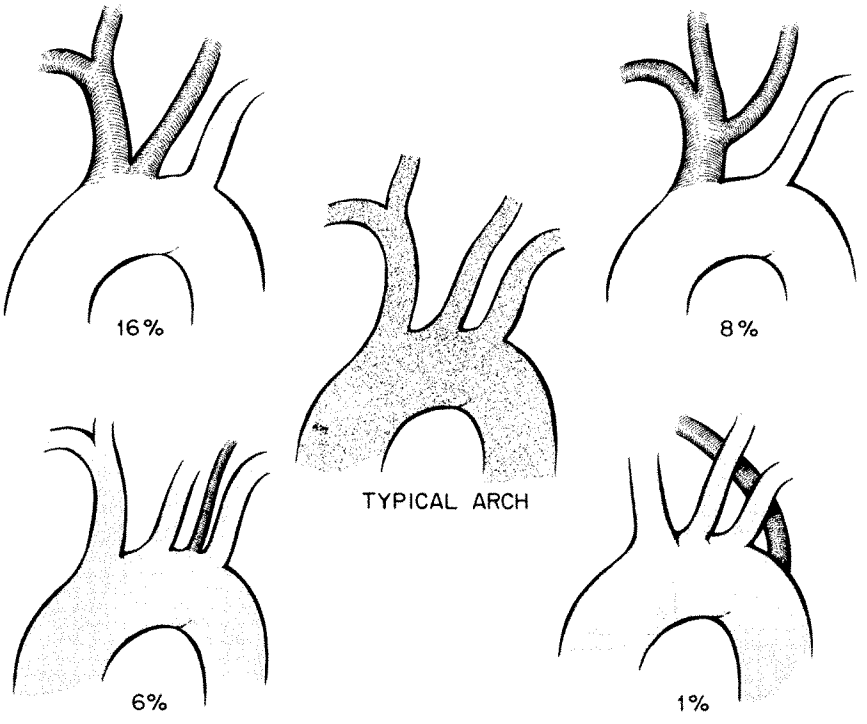


FIG. 1. The most common variations of the branches of the aortic arch with their incidence in the present series.

the brachial approach. There were no deaths or serious complications in this series of cases.

COMMON ANOMALIES OF BRANCHES FROM THE AORTIC ARCH

The usual configuration of 3 branches from the aortic arch is found in 60 to 80 per cent of the population,[1,5,8,9,13,20,21] depending on the racial distribution of the case material. However, as few as 1 branch and as many as 6 vessels arising independently from the arch of the aorta have been described.[6] The common anomalies are shown in Figure 1. In this series, the most common variation was the close association of the origins of the innominate and left common carotid arteries. In 16 patients these 2 vessels were intimately associated, with the left common carotid artery apparently arising at the junction of the innominate artery with the aorta (Fig. 2*B*). In 8 other patients, the left common carotid artery originated entirely from the innominate artery so that this vessel could be considered a common trunk (Fig. 2*A*). This latter configuration is the "prevailing mammalian arrangement of aortic stems" (De Garis *et al.*[5]). The second most common anomaly, being noted in 6 per cent of the cases, was the origination of the left vertebral artery directly from the aortic arch. This vessel always arises between the origins of the left common carotid and left subclavian arteries (Fig. 3). Lastly, 1 case of aberrant right subclavian artery was seen (Fig. 4, *A* and *B*). A second case of this anomaly was seen later in the series (Fig. 5) with a slightly different arrangement of the common carotid arteries. Only 1 case in the series showed more than 1 anomaly of the arch—a common carotid trunk as well as an aberrant right subclavian artery (Fig. 4*A*).

The incidence and types of anomalies presented in this series are similar to the findings of most other American studies (case material including both Negro and white patients) (Table 1). De Garis *et al.*,[5] and Williams and Edmonds[21] analyzed

their Negro and white case material separately and noted considerable variations between aortic arch patterns in Negro and white patients (Table 1). The frequent finding of a common origin of the innominate and left common carotid arteries in Negroes was reported in both series (more than 35 per cent). Apparently, the variation in the findings of the various studies is due to the varying number of Negroes and whites in the case material (Table 1). The incidence and types of other aortic arch variations were found to be essentially equal in both races.[21]

It is important for those performing arteriography to know these variations in the branches of the aortic arch. Selective catheterization of the left common carotid artery when it is associated with the innominate artery can be quite difficult and in most cases impossible. That this variation occurs in as high as 20 to 30 per cent of the population indicates the frequency with which such a problem may occur. This association of the two carotid arteries at their origin explains why visualization of the left common carotid occasionally occurs when a study of the right-sided brachiocephalic vessels is made by using a right retrograde noncatheter brachial injection. Scott *et al.*[15] report that this occurred in 20 per cent of their cases, corresponding, therefore, to the above mentioned figures concerning the anatomic association of the carotid arteries at their origins.

It must be remembered by those interested in studying the left vertebral artery via the left subclavian artery catheterization technique or via retrograde noncatheter pressure injection in the left brachial artery that nonvisualization of the left vertebral artery at its usual place in the subclavian does not necessarily mean that the vessel is absent or occluded. It can

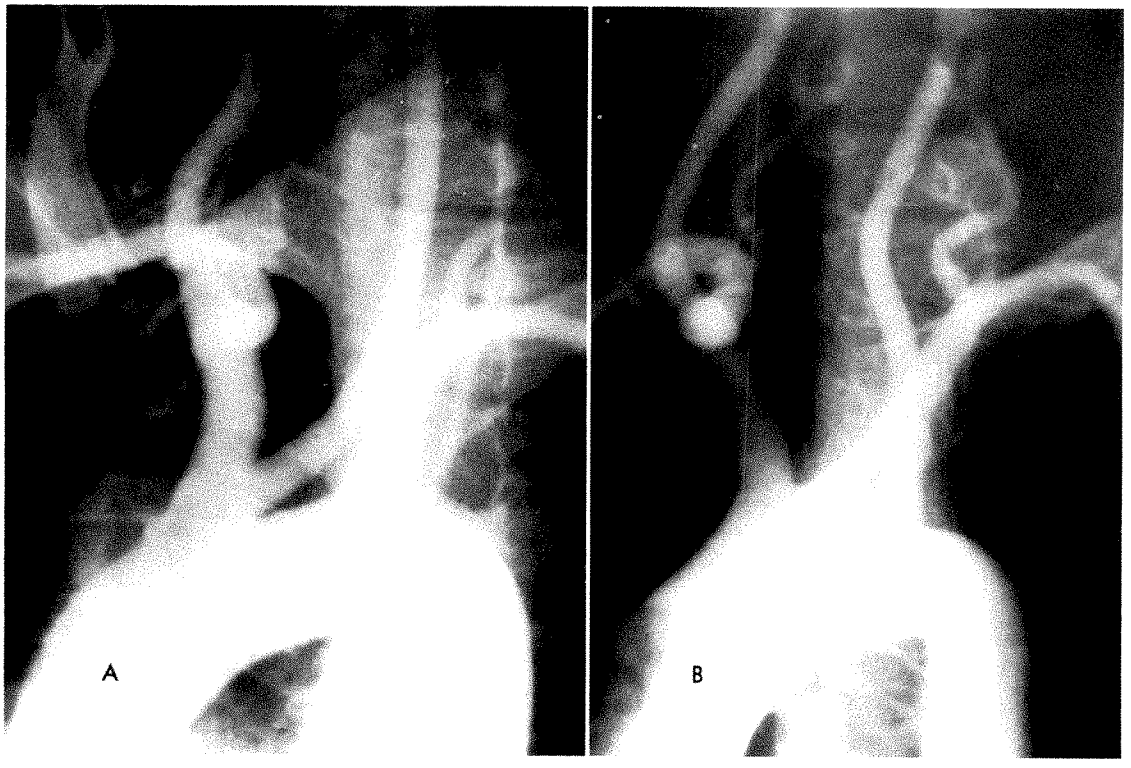


FIG. 2. Roentgenograms showing innominate left common carotid artery variations. (*A*) Left common carotid arises entirely from innominate artery. (*B*) Left common carotid arises from the junction of the innominate artery and aorta.

originate directly from the aortic arch, in which case it would not be visualized by these methods. In Table I, the frequency of the left vertebral artery origination from the aortic arch as found in various series is also shown.

COMPARATIVE SIZE OF THE VERTEBRAL ARTERIES

Total brachiocephalic arteriograms permitted an accurate comparison of the size of the vertebral arteries. The frontal view was used when taking the measurements and the vertebral arteries were measured at the level of C5. There are many autopsy studies concerning vertebral artery size;[2,12,17,19] however, measurement of the lumen of these vessels as determined by arteriography is perhaps somewhat more meaningful (Table II). In 45 cases of the present series, the size of the vertebral arteries was equal or very near equal (not more than 0.5 mm. difference in lumen diameter) so that flow of contrast medium appeared essentially to be the same. There was considerable variation in the "normal" vertebral artery size but the lumen of most

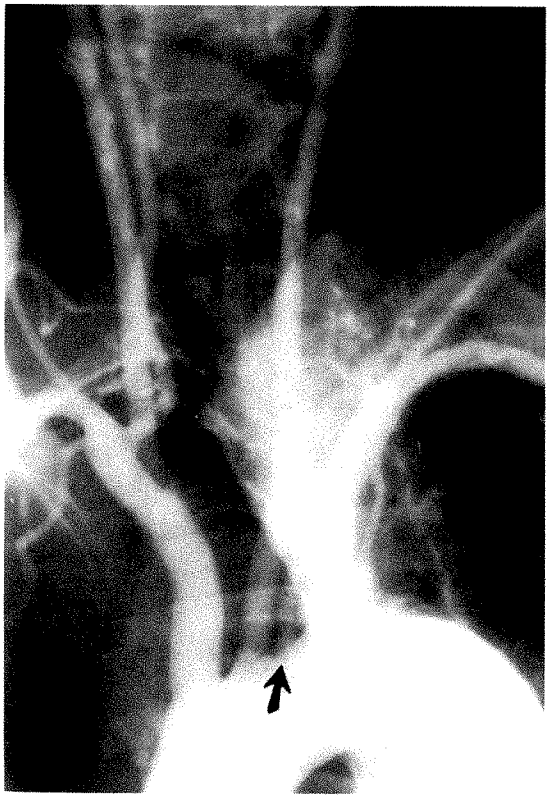


FIG. 3. Left vertebral artery originates directly from aortic arch (arrow).

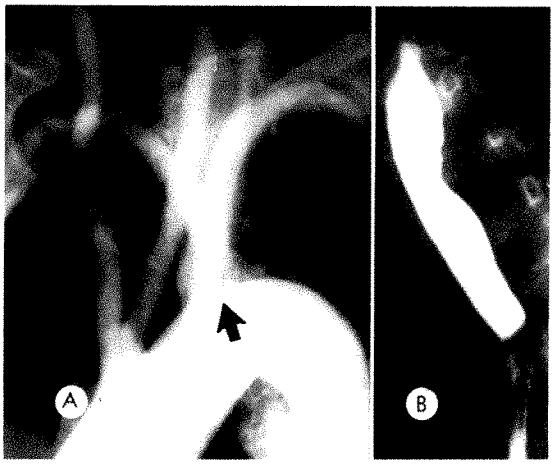


FIG. 4. Aberrant right subclavian artery. (*A*) Aberrant vessel (arrow) courses behind other branches and then gives off right vertebral artery in a normal position. Note common trunk of right and left common carotid arteries. (*B*) Note characteristic impression on the posterior surface of the barium filled esophagus caused by this anomalous vessel.

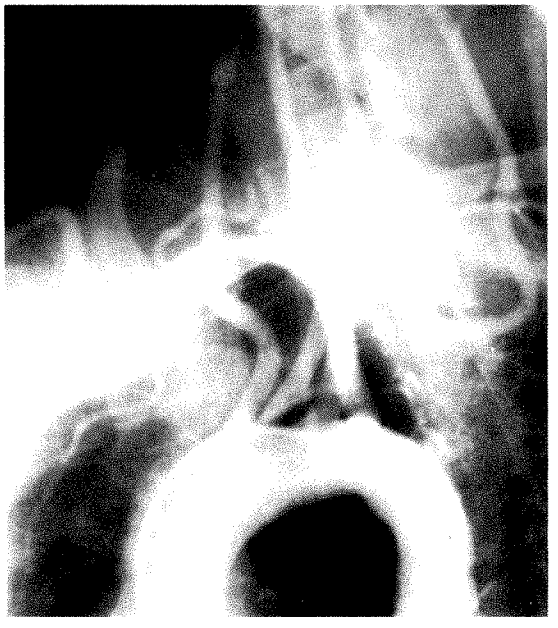


FIG. 5. Aberrant right subclavian artery. Four separate vessels are seen originating from the aortic arch.

TABLE I

INCIDENCE OF THE COMMON VARIATIONS OF BRANCHES OF THE AORTIC ARCH

Authors	No. of Cases	Type of Material	Typical Arch (per cent)	Common Origin of Innominate and Left Common Carotid Artery (per cent)	Left Vertebral Artery Originating from Arch (per cent)	Aberrant Right Subclavian Artery (per cent)
De Garis *et al.*[5]*	111 (Whites) 203 (Negroes)		82.0 51.2	13.5 35.4	2.6	1.6
	314 Total	Autopsy	62.1	27.7		
Williams and Edmonds[21]*	191 (Whites) 216 (Negroes)		75.9 56.9	17.8 37.0		
	407 Total	Autopsy	66.1	28.0	4.2	1.7
Liechty *et al.*[8]*	1,000	Autopsy	64.9	29.0	3.6	1.3
Martin *et al.*[9]	100	Autopsy	68.0	25.0	7.0	
Thomson[20]*	500	Autopsy	82.0	11.0	6.0	1.0
Radner[14]	100	Autopsy			6.0	
Present Series	100	Roentgenography	69.0	24.0	6.0	1.0

* Consolidation of some arch patterns performed to conform with this general classification.

TABLE II

COMPARATIVE SIZE OF VERTEBRAL ARTERIES

Authors	No. of Cases	Type of Material	Right Greater Than Left (per cent)	Left Greater Than Right (per cent)	Equal (per cent)	Right Hypoplastic (per cent)	Left Hypoplastic (per cent)
Stopford[17]	150	Autopsy	41	51	8	7.5	7.5
Compilation of Literature to 1916 (Stopford[17])	301	Autopsy	16.2	21.6	62.2		
Thomas *et al.*[18]	200	Arteriography	31	46	23		
Thomas *et al.*[19]	77	Autopsy				12.3	4.6
Blackburn[2]	220	Autopsy				9.5	9.5
Present Series	100	Arteriography	25	30	45	5	6
Total*	751		26.2	35.0	38.8		

* Excluding 4 and 5.

of these "normal" vessels measured in the range of 3 to 5 mm. In 21 cases, the right vertebral artery was obviously dominant with a difference in vessel lumen size of at least 1.0 mm. In 24 cases, the left vertebral artery was dominant by the same amount. Four cases showed a slightly dominant right vertebral artery (0.5 to 1.0 mm. wider than on the left) and 5 cases showed a slightly dominant left vertebral artery (by the same margin). In 5 cases, the right vertebral artery was hypoplastic (1.5 mm. or less in diameter); in 6 cases the left was hypoplastic (Fig. 6). Absence of the vertebral arteries could not be evaluated because there was no way to determine if the vessel was congenitally absent or occluded at its origin; this was seen in 3 cases.

The findings presented in this series of cases are in general agreement with other surveys in the literature (Table II). It would seem that the left vertebral artery tends to be the dominant vessel slightly more commonly than the right. In 10 to 15 per cent of cases one vertebral artery is hypoplastic. In addition, Neimanis[10] noted congenital absence of a vertebral artery in 7.2 per cent of 418 cases. In view of this, the importance of doing total brachiocephalic arteriography when diagnosing and treating cerebrovascular disease becomes more apparent. Thomas *et al.*[19] point out the importance of knowing the status of both vertebral arteries prior to surgery when operations in which the left subclavian artery is to be used and the left vertebral artery is to be sacrificed (*e.g.*, Blalock operation, surgery for coarctation of the aorta) are performed. These authors described a case of a young man who died following cardiac surgery in which the left vertebral artery was ligated. The right vertebral artery was hypoplastic and the circle of Willis was anomalous; therefore, posterior cerebral circulation was cut off.

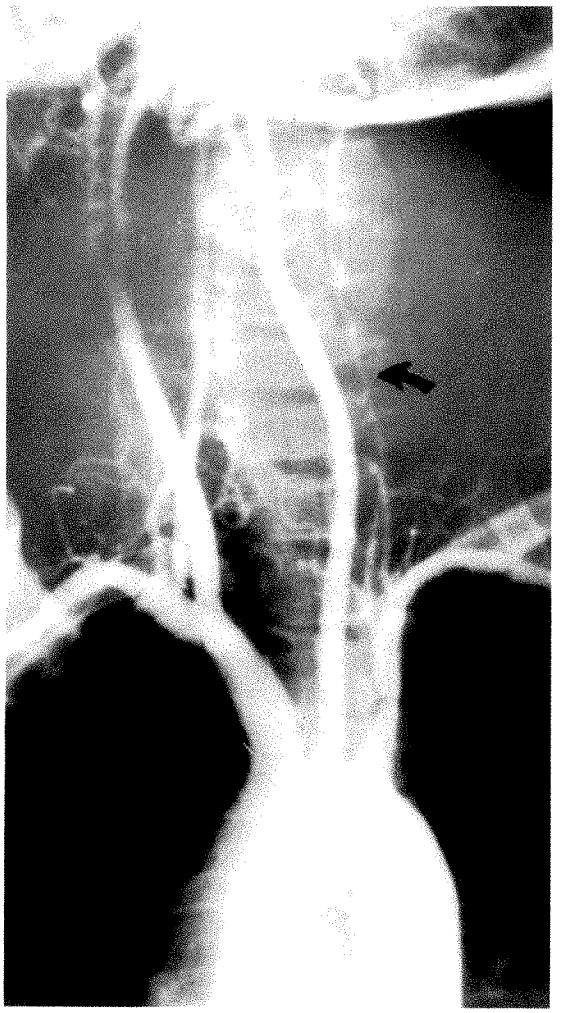


Fig. 6. Hypoplasia of the left vertebral artery (arrow).

Effect of Age and Blood Pressure on the Aortic Arch

The extent to which uncoiling of the aortic arch increased with age and elevation of blood pressure was also noted in this series of cases. The position and direction of the origin of the left subclavian artery with respect to the arch were utilized as a rough guide in evaluating this aortic arch elongation. Since there is a normal anatomic variation in the position of origin of this vessel,[5] the results cannot be interpreted statistically but rather in a general fashion.

It was clearly noted that in patients of the age of "55 and over" the left subclavian artery tended to originate from the arch of the aorta in an anterior position and usually at a slight angle with respect to the top of the arch so that a straight catheter, passed into the arch through this vessel, would invariably course toward the right

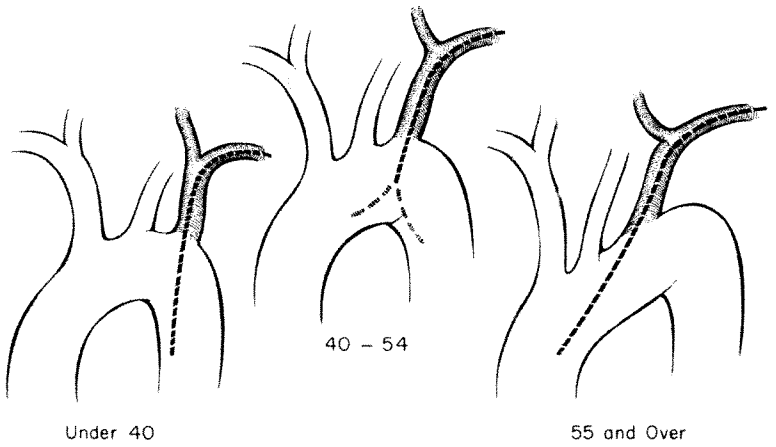


FIG. 7. Changes of the arch with increasing age. The catheter readily enters the descending aorta in the "under 40" arch and enters the ascending aorta in the "55 and over" arch.

and anteriorly and enter the ascending aorta (Fig. 7, and 8*A*). Conversely, in those patients "under 40," the left subclavian artery originated vertically and from the very top of the arch of the aorta so that a straight catheter would enter the descending aorta via the left subclavian artery (Fig. 7, and 8*B*). Patients in the age group "40–54" showed considerable variability. They usually fell between the above two

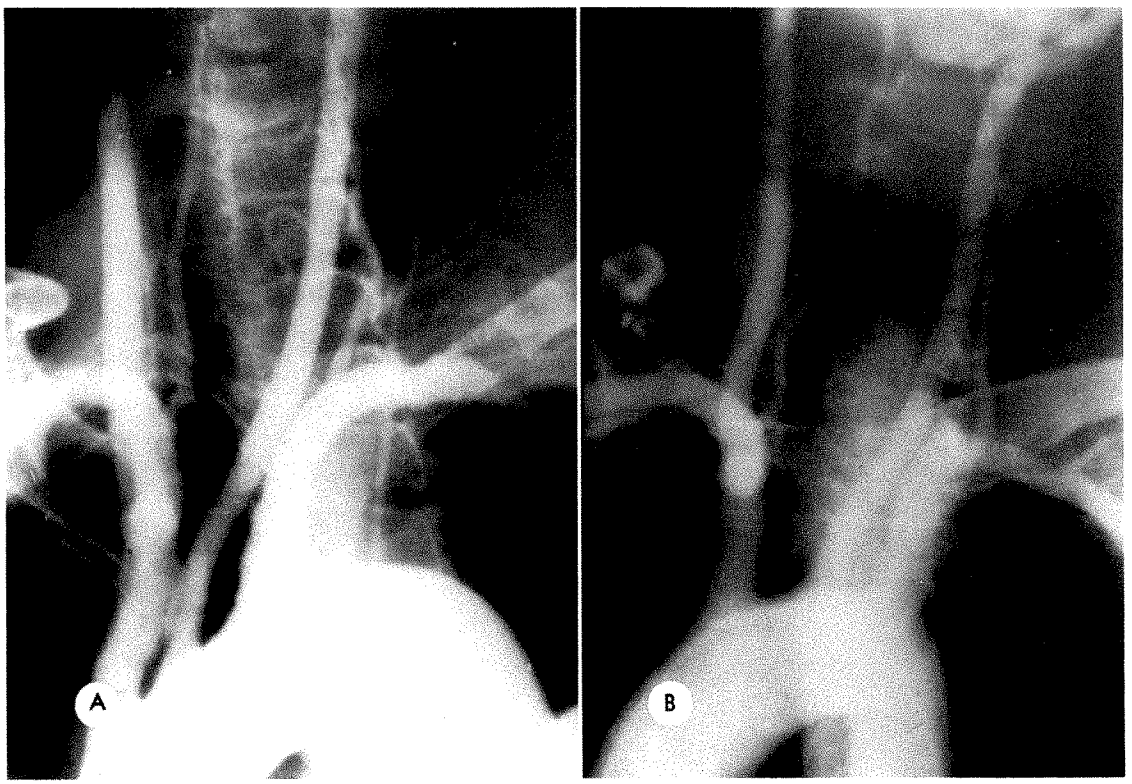


FIG. 8. The effect of age on the aortic arch is shown. (*A*) Sixty-five year old patient. Note anterior position of origin of left subclavian artery. (*B*) Thirty-nine year old patient. Note that left subclavian artery arises from top of arch and is relatively perpendicular to arch.

groups so that one could not be certain from the aortogram whether or not a catheter passed through the left subclavian artery would enter the ascending or descending aorta (Table III). However, patients in the "40–54" year age range, with hypertension or evidence of accelerated arteriosclerosis, would inevitably fall in the "above 55" aortic arch pattern (Fig. 9). Prolonged elevation of blood pressure tends to uncoil the aorta considerably so that the most pronounced "over 55" aortic arch patterns were noted in hypertensives. This series included a large number of such patients.

The practical importance of these findings becomes even more obvious in relation to catheterization procedures. When catheterization of the aortic arch is contemplated using the femoral approach, the necessary curvature of the end of the catheter can be estimated by the patient's age and blood pressure. In patients in the younger age group ("under 40"), a straight catheter passed up from below will tend to enter the left subclavian artery (and left vertebral artery), making the study of these vessels from this approach relatively simple. If the catheter tip is to be placed into the ascending aorta, a sharp curve must be placed near the tip to enable the catheter to make the curve and turn into the ascending aorta without hooking onto the subclavian artery. Conversely, in the older age group, a more gentle curve can be utilized because the catheter is less likely to enter the left subclavian artery.

In the older age group, a straight catheter can be utilized in arch aortography via the brachial or axillary approach from the left side, as the catheter invariably will enter the ascending aorta. We utilize the left-sided approach in this group because the catheter bypasses the innominate artery. In older patients, it may be difficult to pass a catheter through the innominate artery because frequently this vessel is buckled and tortuous. If the ascending aorta is to be catheterized in the "under 40" group, the right side should be used because the catheter tends to go down into the descending aorta when done via the left side. To enter the descending aorta in the older age group, a curved catheter must be used via the left subclavian artery approach. If desired, a straight catheter can then be placed in the descending aorta by inserting a wire into the curved catheter that is already in the descending aorta, removing the

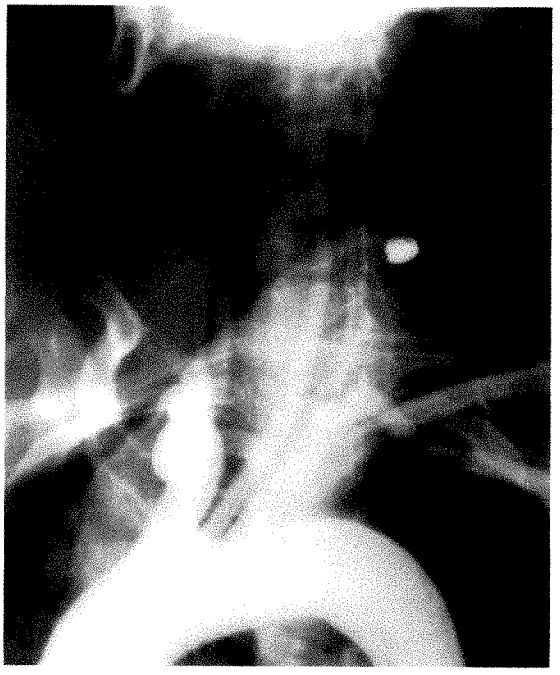


Fig. 9. The effect of increased blood pressure is demonstrated. Forty year old female with hypertension for 10 years. Blood pressure at time of examination was 240/150 mm. Hg. Note considerable uncoiling of arch tending to "over 55" arch pattern. Left subclavian artery is anteriorly placed and originates from the arch at approximately a 45 degree angle.

Table III

Effect of Age on Aortic Arch

Age Range	No. of Cases	Direction of Catheter Via Left Subclavian Artery		
		Ascending Aorta	Descending Aorta	Uncertain
55 and over	56	52	2	2
40–54	35	17	7	11
Under 40	9	0	8	1

curved catheter and then passing the straight catheter over the wire.

Cronqvist[4] found, in a study of the vertebral artery by the transfemoral approach, that he could easily place the catheter into the left vertebral artery in patients under 40 years of age (92 per cent) but in less than 50 per cent of the patients over the age of 40.

Generally, it is quite simple to catheterize the vertebral artery by the transbrachial (or axillary) approach, particularly in the older age group as the vertebral artery frequently buckles slightly in its proximal portion. This tends to make the mouth of this vessel more accessible to a catheter passed in such a manner (Fig. 7). Radner[14] found that a catheter passed via the radial artery entered the vertebral artery on the first try in 70 per cent of his cases. With some manipulation, he was able to pass the catheter successfully into the vertebral artery in the remainder of the cases. Radner[14] and other writers[3,7,11] suggest a right-sided approach because of the more constant relationship of the right vertebral artery to the subclavian artery. Thus, it appears that vertebral artery catheterization studies can be more easily done by using the femoral approach in the "under 40" group and the right transbrachial or axillary approach in those over this age.

SUMMARY

An analysis of some aspects of the roentgenologic anatomy, common anomalies, vertebral artery size, and effect of age on the aortic arch and branches was made of 100 total brachiocephalic arteriographies. A comparison with other series is included.

The high incidence of the association of the origin of the innominate artery and the left common carotid artery (24 per cent) was confirmed. Origin of the left vertebral artery from the aortic arch was noted in 6 per cent of the patients. A comparison of vertebral artery size was also made and a slight predominance of the left vertebral artery was noted.

The importance of these factors along with the effect of changes in the contour of the aorta with age and blood pressure is discussed as regards catheterization techniques.

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The author would like to thank Drs. Irwin Entel and Charles Walkoff for performing some of the arterial studies included in this series.

ADDENDUM

An analysis of the second 100 total brachiocephalic arteriograms in our series revealed anatomic-roentgenologic findings that were similar to those described for the initial 100 cases included in this report.

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CERVICAL ARTERIAL PATHWAYS ASSOCIATED WITH BRACHIOCEPHALIC OCCLUSIVE DISEASE*

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DURING the past few years the roentgenographic demonstration of collateral arterial pathways has been enhanced by the increased use of total brachiocephalic arteriography and the more widespread employment of rapid serial film changers. This has enabled the radiologist to visualize and evaluate the importance of many collateral arterial pathways not previously known or appreciated. One example is the recent discovery and demonstration of the reversal of blood flow in the vertebral artery associated with innominate and proximal subclavian artery obstruction ("subclavian steal").[3,5,8,20] A rich collateral arterial anastomosis, frequently utilized in brachiocephalic occlusive disease, exists in the neck which connects the carotid, vertebral and subclavian arteries. These pathways, which have been termed the "cervical arterial collateral network" form the basis of this report.

Total brachiocephalic arteriography has been performed on more than 150 patients at the Montefiore Hospital, New York, during the past 2 years. An analysis of some aspects of the radiologic anatomy of these cases as well as a discussion of techniques used in the performance of these studies has been reported.[4] The great majority of the studies were done on patients with symptoms of cerebrovascular insufficiency to evaluate extracranial occlusive disease. The number and types of collateral arterial pathways encountered in occlusive disease of the brachiocephalic vessels are many. No attempt will be made to discuss all of the collateral channels but only the more common ones. Of these, collateral circulation through the circle of Willis is the most important and most frequently encountered collateral pathway in occlusive disease of the extracranial cerebral circulation. Cross-over filling via the circle of Willis in cases of occlusive disease of an internal carotid artery is the most important and frequently observed pathway. In these cases, blood flow to the carotid siphon on the affected side occurs via the anterior communicating artery from the contralateral carotid artery (Fig. 1, *A* and *B;* Case 1).[14,16,27,33] In occlusive disease of both vertebral arteries or of the basilar artery, filling of the posterior circulation by the internal carotid arteries via the posterior communicating vessels of the circle of Willis can occur (Fig. 2, *A* and *B;* Case II).[13,14,22,24] Likewise, in bilateral internal carotid occlusive disease, filling of the anterior circulation by the vertebral arteries through the posterior communicating arteries is occasionally observed (Fig. 3, *A* and *B;* Case III).[9,14,22,30] Occasionally, associated with the above pathways but generally independent is collateral circulation through the external carotid artery to the ipsolateral internal carotid (at the siphon) via the ophthalmic artery (Fig. 4; Case IV).[7,11,12,25,32]

The reversal of blood flow in the vetebral artery associated with innominate and proximal subclavian artery occlusion, *i.e.*, the "subclavian steal" has been found to be another very important and common collateral pathway. In most instances the contralateral vertebral artery supplies the occluded vessel by way of the vertebrobasilar junction with retrograde flow down the vertebral artery on the involved side.[2]

This author has noted the occasional occurrence of a group of collateral path-

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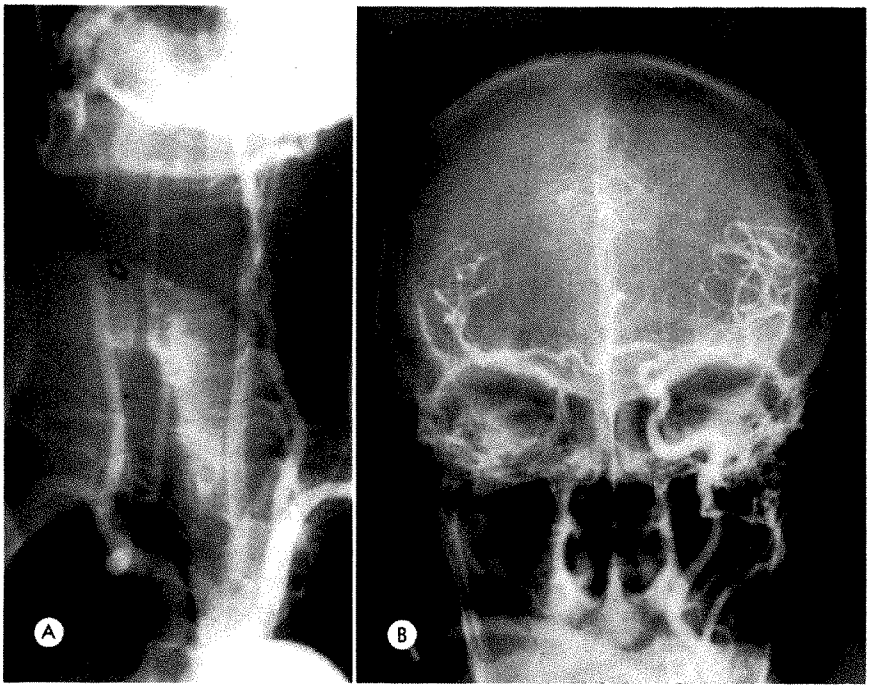


FIG. 1. Case I. (*A*) Arch aortogram shows complete occlusion of right internal carotid artery at its origin (arrow). (*B*) Selective left common carotid arteriogram reveals "cross-over filling" to right-sided cerebral vessels by way of anterior communicating artery of circle of Willis.

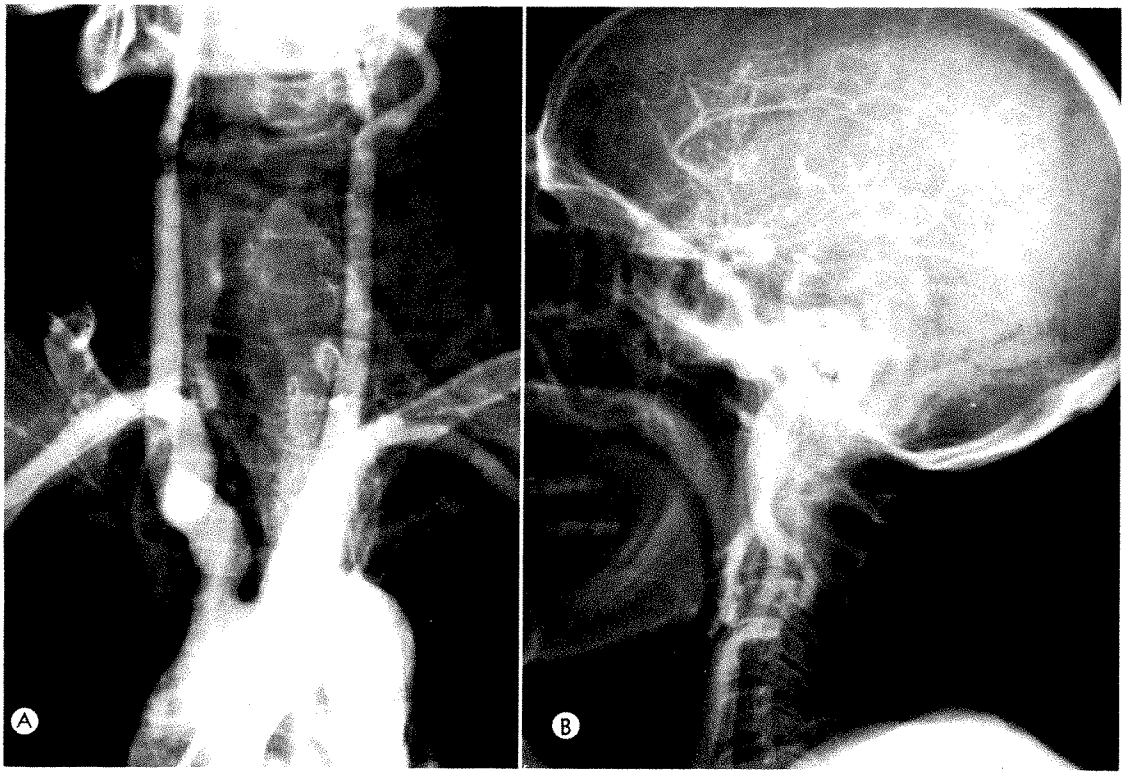


FIG. 2. Case II. (*A*) Arch aortogram reveals complete occlusion of proximal right vertebral artery (arrow) and nonvisualization of left vertebral artery. Note, also, narrowing at origin of left internal carotid artery. (*B*) Cerebral portion of study reveals filling of distal basilar artery and posterior circulation entirely by both internal carotids via posterior communicating arteries of circle of Willis. No vertebral artery filling is apparent.

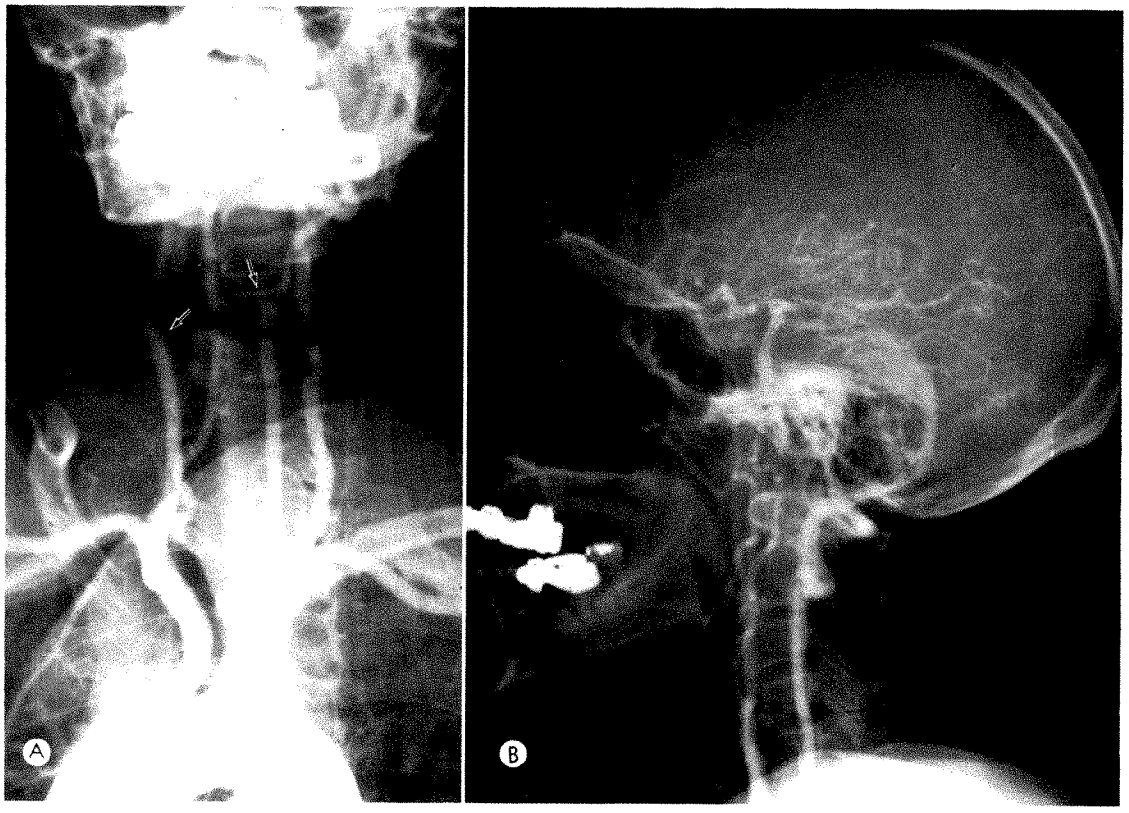


FIG. 3. Case III. (*A*) Complete occlusion of both internal carotid arteries at their origins (arrows) can be seen. Note large size of vertebral arteries particularly on the left side. (*B*) Filling of anterior circulation can be seen by way of the posterior communicating arteries of the circle of Willis from an enlarged basilar artery. Some filling of both siphons is probably also being supplemented by flow through the ophthalmic arteries.

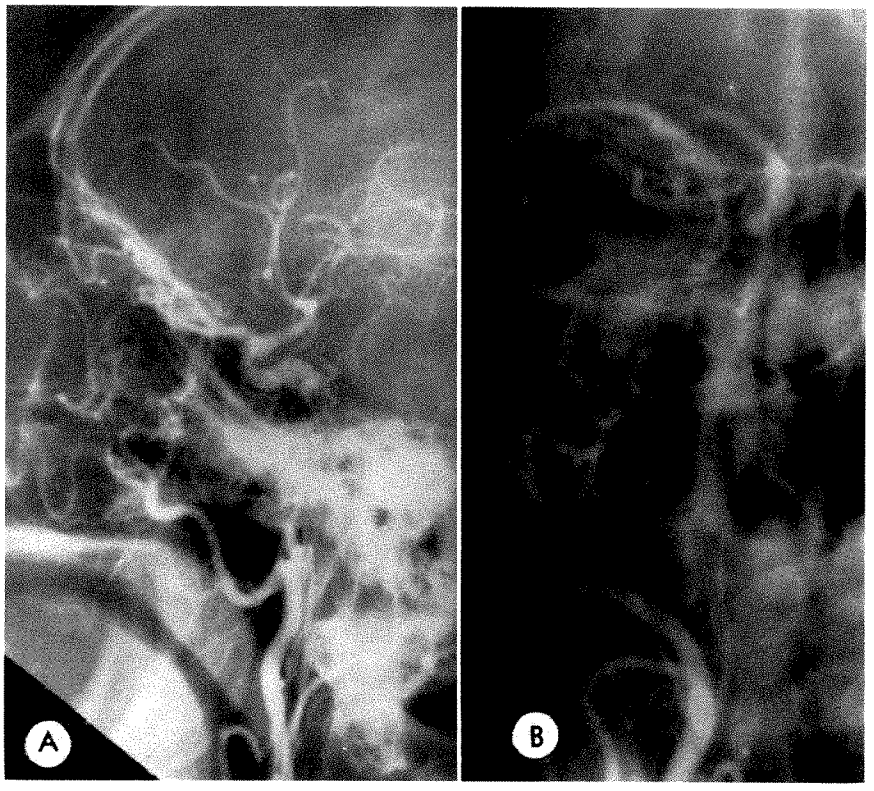


FIG. 4. Case IV. (*A*) Lateral and (*B*) anteroposterior roentgenograms show complete occlusion of the internal carotid artery at its origin with filling of the carotid siphon entirely by the external carotid artery via anastomosis with the ophthalmic artery about the orbit.

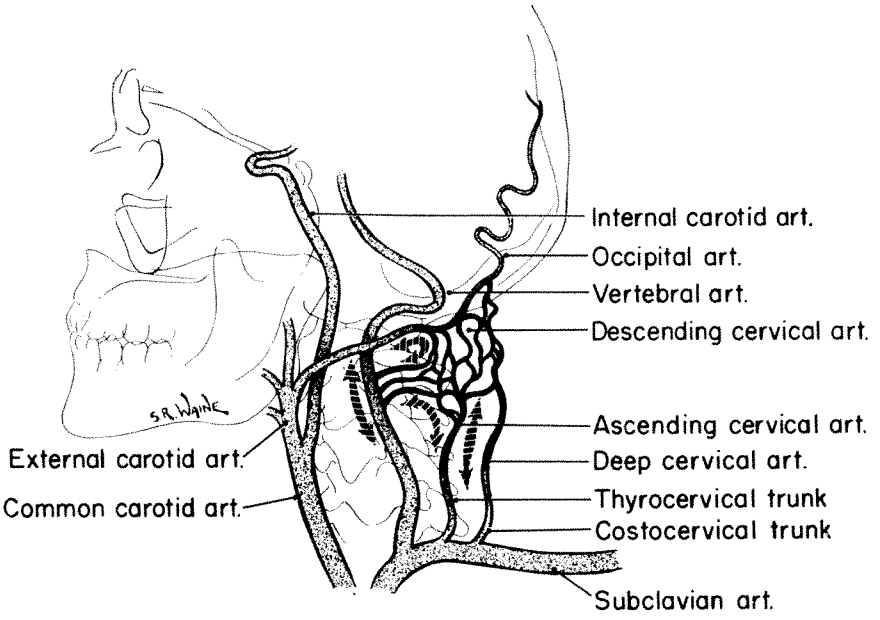


Fig. 5. The "cervical arterial collateral network" which joins the carotid, subclavian and vertebral arteries.

ways that link the carotid, subclavian and vertebral arteries. This has been referred to as the "cervical arterial collateral network." This network utilizes the occipital branch of the external carotid artery, the muscular branches of the vertebral artery and the thyrocervical and costocervical trunks of the subclavian artery (Fig. 5). In some cases of occlusive disease, flow through this network may be the sole or major blood supply. Frequently, these channels may be used in combination with other collateral pathways to supplement blood supply.

These anastomoses are well known to most anatomists.[6,10,19] With the increased use of arteriography and serial roentgenography, these channels have been roentgenographically visualized and this has enabled a more complete understanding of these pathways anatomically and, more important, hemodynamically. A knowledge of the available collateral channels is necessary before operative repair of occlusive disease can be undertaken. Table 1 and Figure 6 show that there are 7 possible pathways through this network. It can be seen that the same pathway can be utilized in each direction, *e.g.*, the same pathway is used in occlusive disease of the subclavian artery (pathway 1a) as well as in occlusive disease of the external carotid artery (pathway 2a). In any particular case, whether or not they will become important collateral channels depends on the site of the obstruction and the condition of the other collateral pathways. Some members of this group are rather common (1a) while others are quite rare (3a, 1b).

In proximal subclavian artery occlusion, the reversal of flow in the ipsolateral vertebral artery with supply from the contralateral vertebral artery ("subclavian steal") would appear to be the easiest and largest collateral pathway to be utilized. It is already present and requires only a drop in pressure in the subclavian artery to reverse the flow through the vertebral artery.[20] However, if for some reason this pathway cannot be used (occlusive disease or hypoplasia of either vertebral artery) then pathways 1a or 1c (Table 1; Fig. 6) would be utilized, depending on local conditions. Pathway 1c was recently described by Simon *et al.*[29] and Philp *et al.*[18] as one of the pathways of the "subclavian steal." In this

type of "steal" pattern, the homolateral external carotid artery supplies the subclavian artery via retrograde flow down the vertebral artery. Pathway 1a has also been observed in occlusive disease of the proximal subclavian artery when the vertebral artery was found to be occluded (Fig. 7, *A* and *B;* Case v). When subclavian artery occlusion is distal to the origin of the vertebral artery, then pathway 1a through the thyrocervical and costocervical trunks may be most important (Fig. 8, *A* and *B;* Case vi). Frequently, this pathway is noted supplementing other pathways in subclavian artery occlusive disease.[2,3,15,18]

Filling of the distal subclavian artery via the muscular branches of the vertebral and the cervical trunks can also occur as shown in pathway 1b. A case of bilateral congenital aplasia of the proximal subclavian arteries with associated coarctation of the aorta is shown in Figure 9, *A* and *B;* (Case vii). The distal subclavian arteries (and upper extremities) were supplied to a great extent by large muscular branches of the vertebral arteries via the cervical trunks. This can be considered a congenital "subclavian steal" (Fig. 9, *C* and *D;* Case vii). Other collaterals found in subclavian artery occlusive disease are pathways through the

TABLE I

THE "CERVICAL ARTERIAL COLLATERAL NETWORK"

Pathway	Occluded Artery	Supplying Artery	Course of Pathway
1a	Subclavian	External Carotid	Via occipital artery, descending cervical branch to ascending cervical and deep cervical branches of thyrocervical and costocervical trunks, respectively (same anastomosis, but opposite direction of flow as 2a)
1b	Subclavian	Vertebral	Muscular branches of vertebral artery to cervical branches of thyrocervical and costocervical trunks (same anastomosis but opposite direction of flow as 3a)
1c	Subclavian	External Carotid	Via occipital artery to muscular branches of vertebral artery and retrograde flow down vertebral artery (same anastomosis as 2b and 3b except retrograde flow in vertebral artery)
2a	Carotid (external, internal, common)	Subclavian	Thyrocervical and costocervical trunks via ascending and deep cervical to descending cervical branch of occipital artery (same anastomosis but opposite direction of flow to 1a)
2b	Carotid (external, internal, common)	Vertebral	Muscular branches of vertebral to muscular branches of occipital artery (same anastomosis but opposite direction of flow as 1c)
3a	Vertebral	Subclavian	Thyrocervical and costocervical trunks via ascending and deep cervical branches, respectively, to muscular branches of vertebral artery (same anastomosis but opposite direction of flow as 1b)
3b	Vertebral	External Carotid	Via occipital artery to muscular branches of vertebral artery and then antegrade flow in vertebral artery (same anastomosis but opposite direction of flow as 2b; same pathway as 1c except antegrade flow in vertebral artery in this pathway)

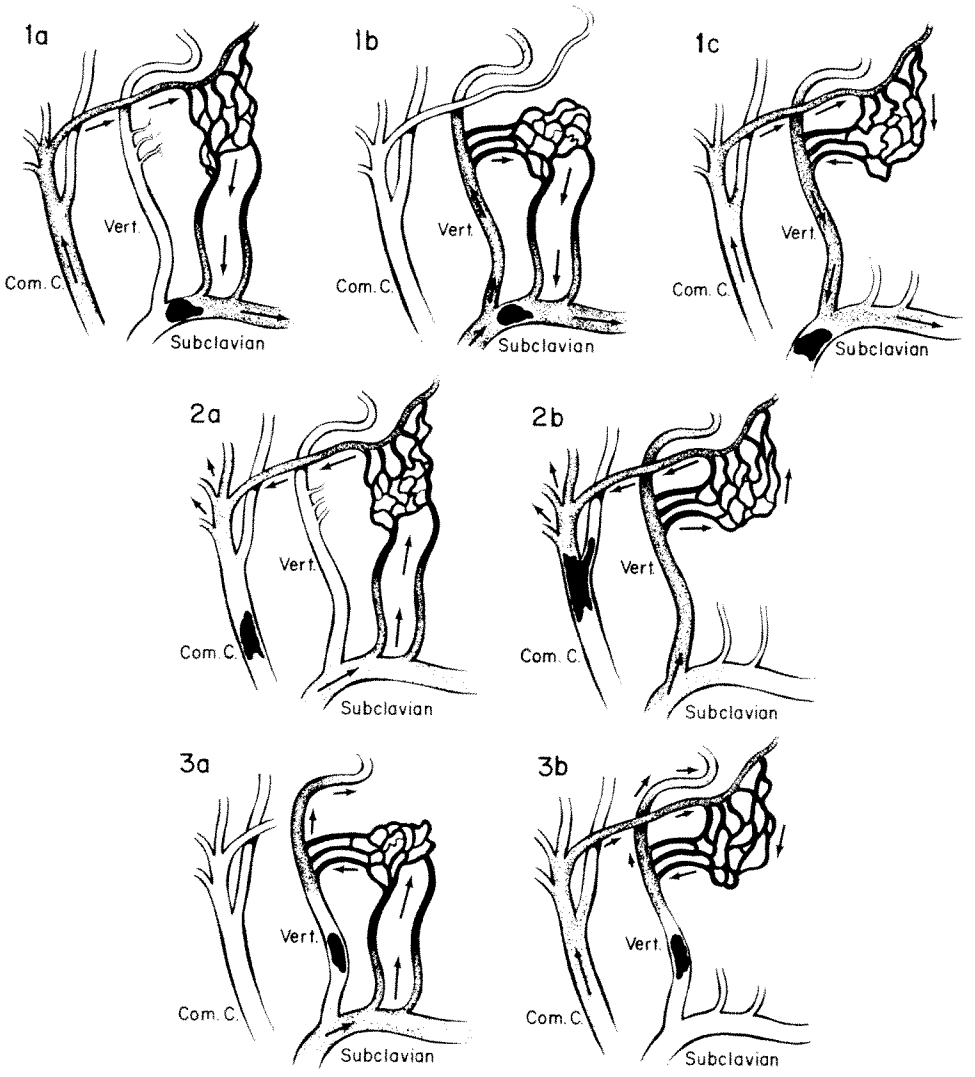


Fig. 6. The 7 possible pathways through the "cervical arterial collateral network" are diagrammed and numbered. (Numbers correspond to description in text and Table 1.)

inferior thyroid arteries via the thyroid circulatory network[15,18,19] and collateral pathways via the internal mammary and intercostal vessels.[15,19] Frequently collateral circulation to the upper extremity is supplemented by flow through the intercostal arteries via the subscapular artery to the axillary artery (Fig. 10).

The ability of the subclavian artery to aid in carotid artery occlusive disease is demonstrated in pathways 2a and 2b (Table 1; Fig. 6; Fig. 11, *A–D*, Case VIII; and Fig. 12, *A–D*, Case IX). In these cases filling of the external carotid artery occurred through different channels of the cervical collateral arterial network. Filling of the internal carotid artery at its origin in occlusive disease of the common carotid artery can also occur through these pathways, as shown in a report by Youmans and Scarcella.[34] Likewise, filling of the internal carotid artery at the siphon could take place through these same pathways by supply to the external carotid artery and then via the ophthalmic artery to the siphon as seen in Case IX (not well demon-

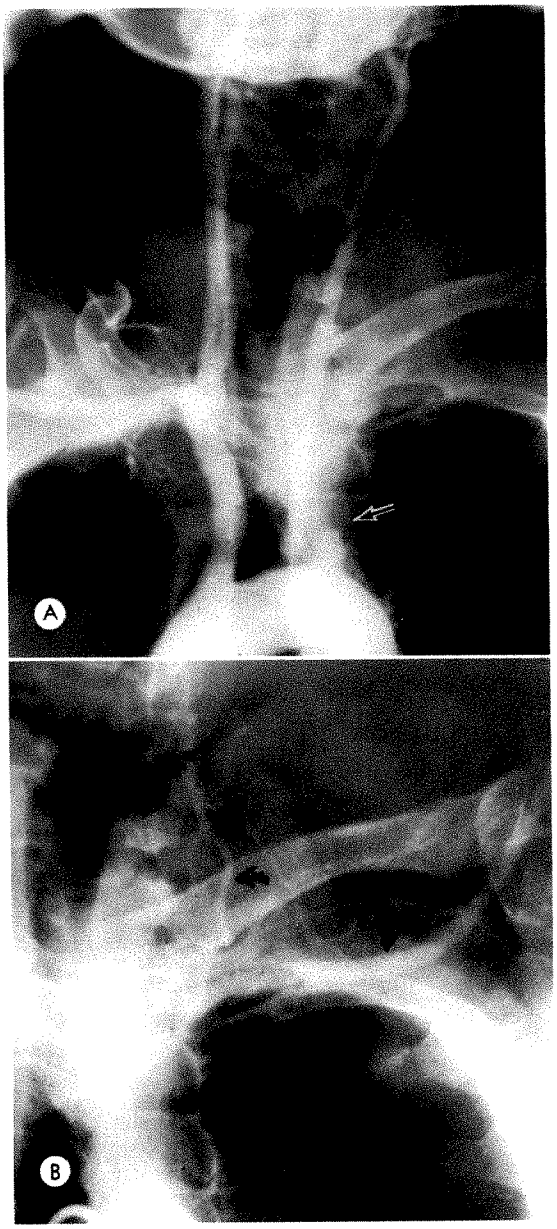


FIG. 7. Case v. (*A*) Arch aortogram demonstrating complete occlusion of the proximal left subclavian artery (arrow). (*B*) Roentgenogram made 2 seconds later. Collateral circulation in this case is almost entirely through pathway 1a via the thyrocervical trunk (arrow). Another arrow points to the opacified left subclavian artery.

strated roentgenographically) (Fig. 12, *A–D*).

The utilization of pathways 3a and 3b (Table 1, Fig. 6) in vertebral artery occlusive disease seems quite remote because occlusion of one vertebral artery can, in most cases, be supplemented satisfactorily and easily by compensatory hypertrophy of the contralateral vertebral artery or via flow through the posterior communicating arteries in the circle of Willis (as previously described). However, if in a given patient these routes of collateral supply to the posterior circulation are not available or insufficient (anomalies of the circle of Willis have been shown to be common),[1,21] then filling of the distal vertebral artery via these pathways in the cervical network will become necessary and perhaps critical.

Pathway 3b in which the vertebral artery is filled via the external carotid artery in occlusive disease of the proximal vertebral artery has been demonstrated roentgenographically by Tatelman.[31] Richter[23] also reported 2 cases in which this pathway was utilized. Other authors have described this anastomosis and mention it as another possible collateral channel in internal carotid artery occlusion.[9,22,28] The presence of this pathway (3b) as the principal supply to the circle of Willis in the ox and horse shows a phylogenetic basis for this communication[26] and an embryologic basis is demonstrated in the work of Padgett,[17] who noted the occurrence of this occipital-vertebral anastomosis in the 4 mm. embryo.

It is expected that more and more of these less well-known collateral channels will be demonstrated as techniques of arteriography continue to improve. Collateral pathways that depend on filling from the opposite side (across the midline) via branches of the external carotid artery have been shown arteriographically in a few instances.[2,15,31] The rich thyroid anastomoses, which connect the subclavian and external carotid arteries on one side as well as these same vessels on the opposite side, will probably prove to be another important supplementary collateral network. Other pathways will be discovered and demonstrated by roentgenography as shown by a recently reported case in which an anas-

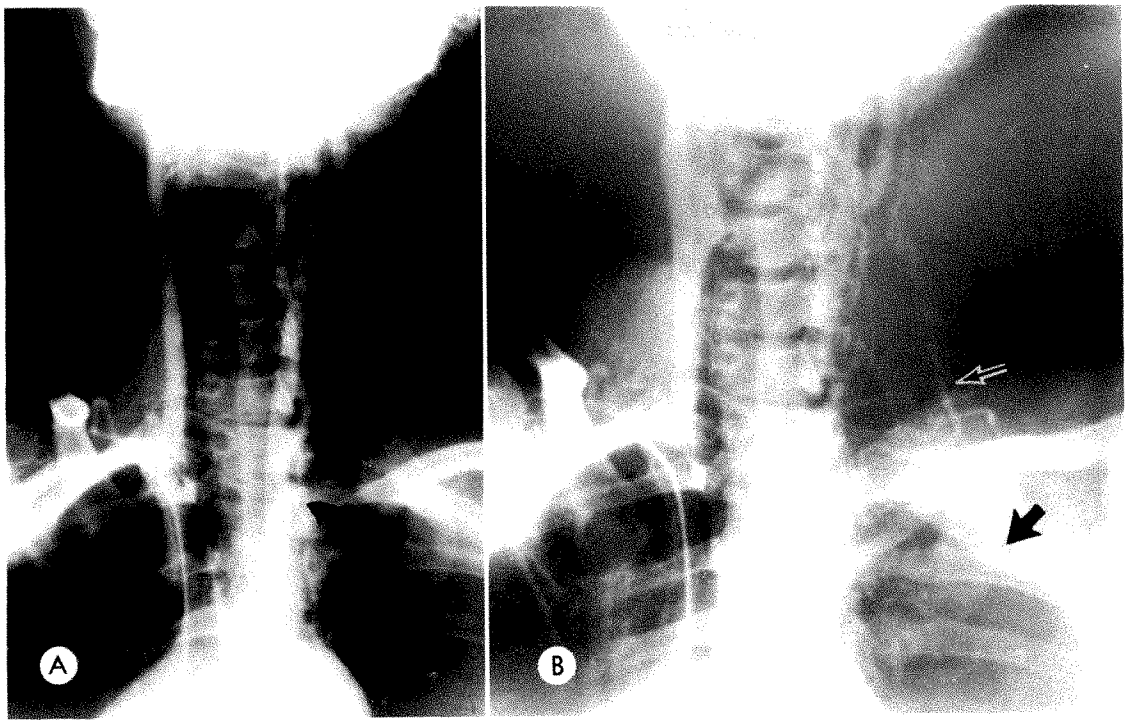


FIG. 8. Case VI. (*A*) Complete occlusion of the left subclavian artery just distal to origin of left vertebral artery (arrow). Note nonvisualization of right vertebral artery. (*B*) Roentgenogram made 1.75 seconds later reveals opacification of left subclavian artery (black arrow) via retrograde filling of thyrocervical and costocervical trunks (white arrow) from occipital artery. Some filling via thyroid collaterals is also present.

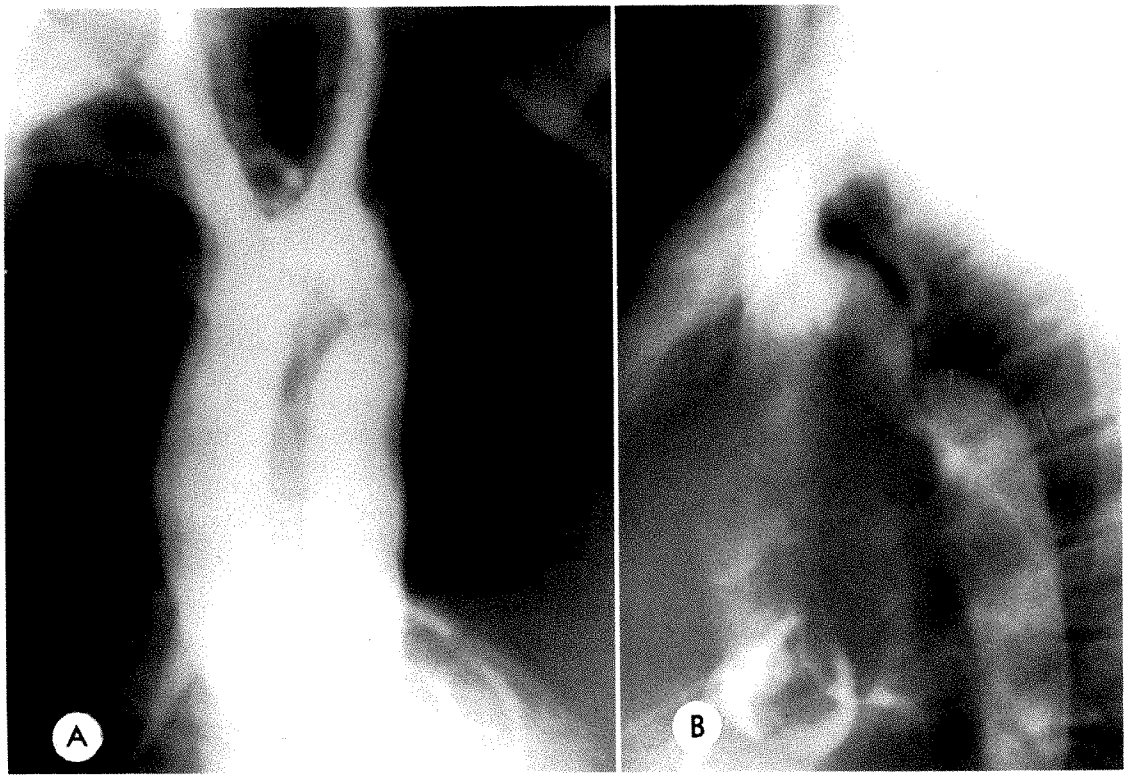


FIG. 9. Case VII. (*A* and *B*) Coarctation of the aorta is demonstrated. Nonvisualization of both subclavian arteries is noted. The left vertebral artery originates from the arch of the aorta just above the coarctation.

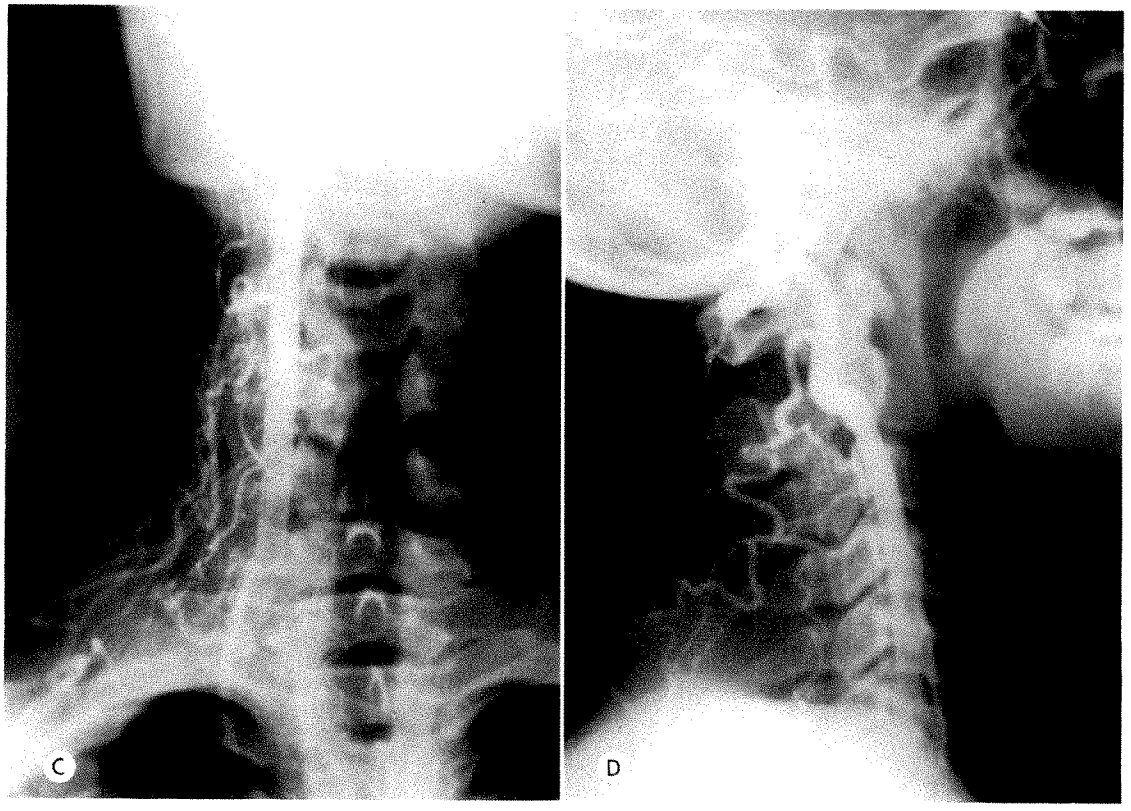


FIG. 9. Case VII. The catheter was then placed into the right vertebral artery which originates directly from the innominate artery. (*C*) Frontal and (*D*) lateral roentgenograms reveal huge muscular branches of the vertebral artery which drain into the cervical trunks to supply the subclavian artery, a congenital "subclavian steal" (pathway 1b).

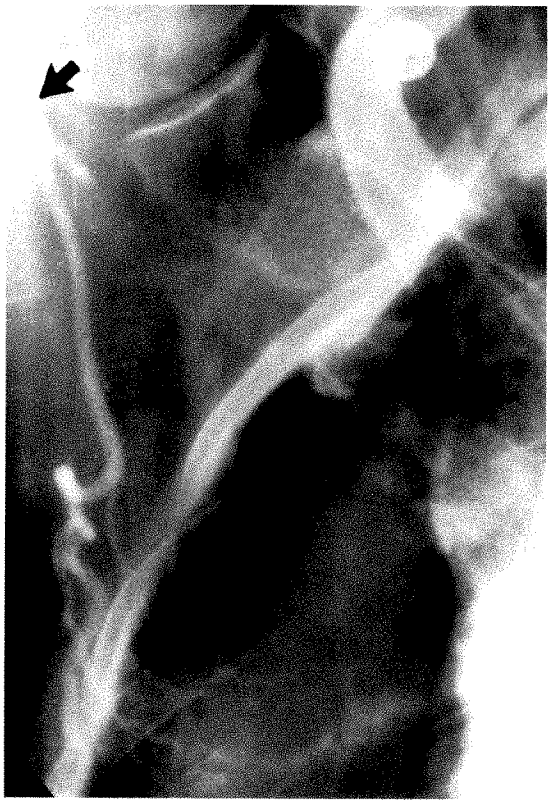


FIG. 10. A pathway which is commonly seen supplementing blood supply to the arm in occlusive disease of the subclavian artery is visualized. Filling of the axillary artery (arrow) by supply from intercostal vessels via the subscapular artery is demonstrated.

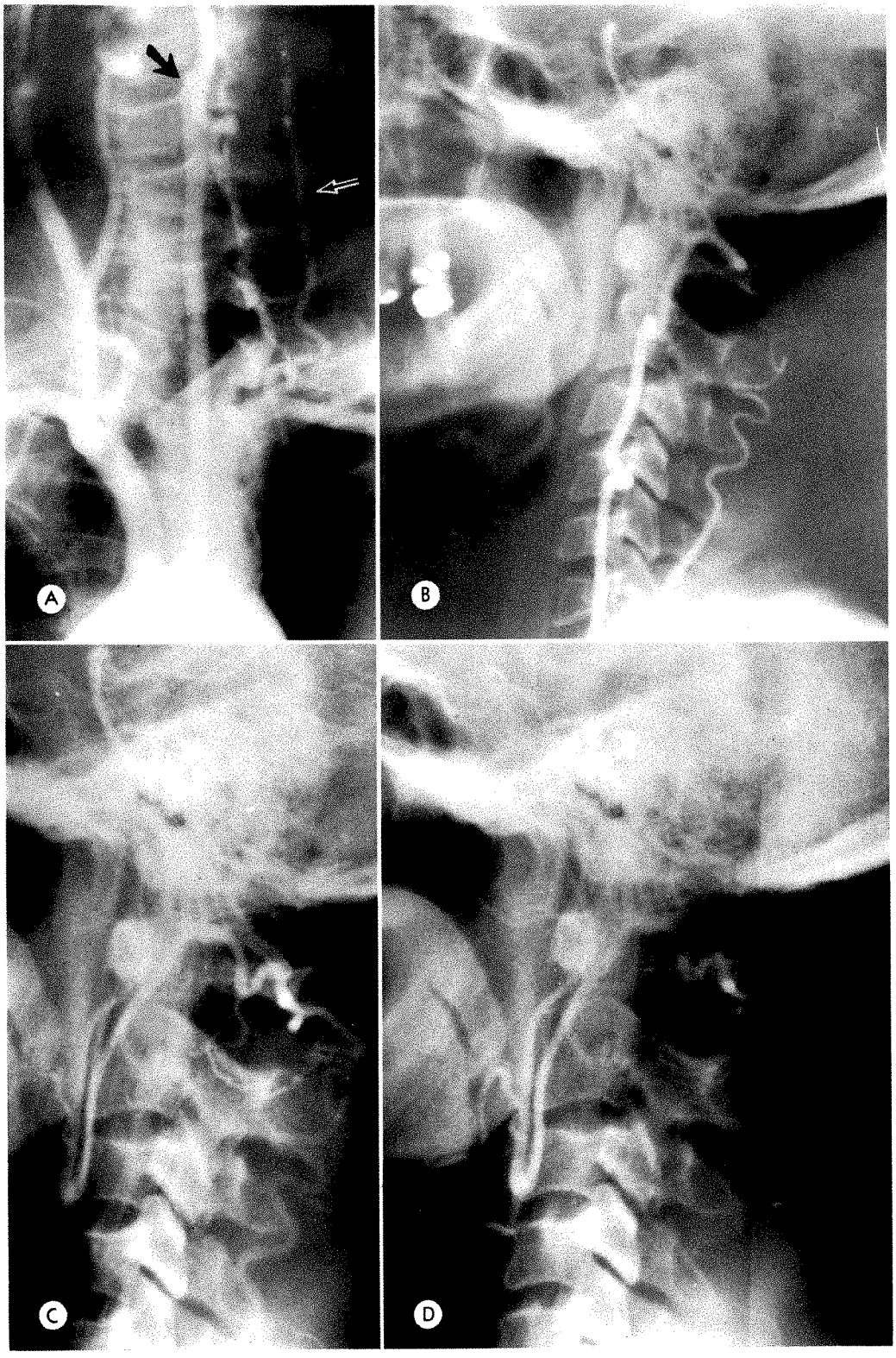


FIG. 11. Case VIII. (*A*) Arch aortogram reveals occlusion of left external carotid artery at its origin (black arrow). Note enlarged ascending cervical branch of thyrocervical trunk (white arrow). (*B*) The catheter was then placed in the left subclavian artery and a second injection was performed. The vertebral artery and basilar artery and its branches are well seen. The enlarged ascending cervical branch of the thyrocervical trunk can be seen posteriorly. A few small muscular branches of the vertebral artery are noted. (*C*) Opacification of the occipital branch of the external carotid artery by filling from the thyrocervical trunk can be seen (pathway 2a). (*D*) Opacification of the external carotid artery and its branches is now more complete.

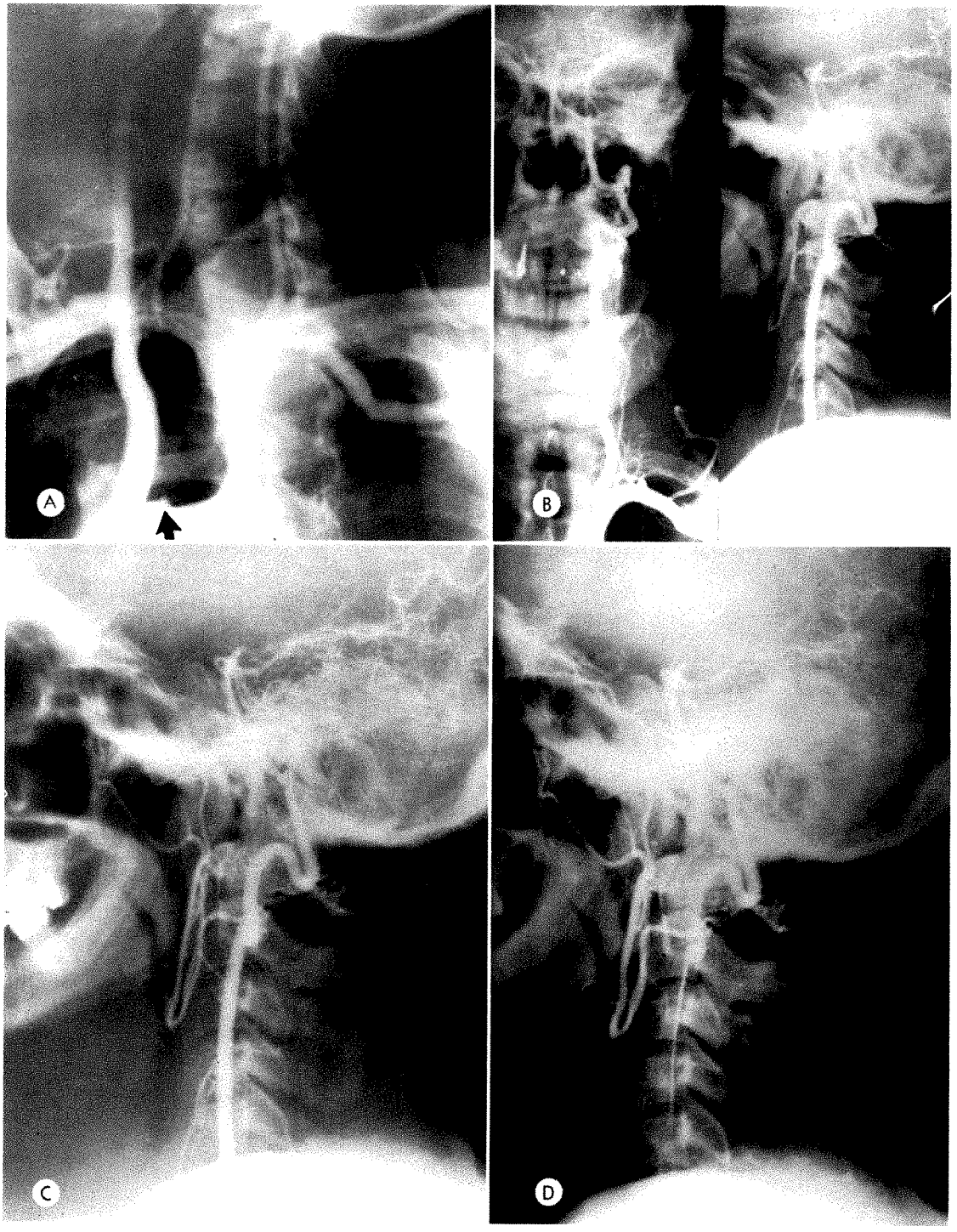


FIG. 12. Case IX. (*A*) Occlusion of left common carotid artery at its origin is demonstrated on the arch aortogram (arrow). Note large size of left vertebral artery. (*B*) The catheter was then placed in the proximal left subclavian artery and a second injection was performed. Opacification of large muscular branches of the vertebral artery and early filling of the occipital branch of the external carotid artery can be seen (pathway 2b). Note that no definite filling of carotid siphon through the postcommunicating vessels has occurred at this time. (*C*) Further opacification of external carotid artery via pathway 2b. (*D*) Complete opacification of external carotid artery and its branches. Note faint filling of carotid siphon through the ophthalmic artery anastomoses about the orbit.

tomosis between the ascending pharyngeal artery (a branch of the external carotid artery) and muscular branches of the vertebral artery occurred in a case of internal carotid artery occlusion.[35]

Almost any combination of pathways can occur in a given case. The well-known pathways are utilized more frequently. When these pathways are unable to supply the demand, any one or multiple pathways can develop depending on the particular anatomy of the individual.

SUMMARY

The major collateral pathways associated with occlusion of the brachiocephalic vessels are reviewed and illustrated. Less known collateral arterial pathways which join the carotid, vertebral and subclavian arteries in occlusive disease of these vessels are also discussed. These anastomoses, termed the "cervical arterial collateral network" are described, diagrammed, and demonstrated roentgenographically.

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OBSERVATIONS ON RETROGRADE VERTEBRAL ARTERY BLOOD FLOW*

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OCCLUSIVE arterial disease, partial or complete, involving the innominate, carotid and subclavian arteries will produce different degrees of disability depending upon the establishment of collateral circulation, the degree of stenosis and the site of stenosis. Angiographic studies of the vessels of the neck and the aorta, using different approaches, have shown in living subjects both the occlusive lesion and the collateral channels, which have developed in this disease.[2,4,5,22]

When there is partial or complete occlusion of a subclavian or innominate artery, depriving the ipsilateral arm of its normal blood supply, this will be corrected in some patients by reversal of blood flow in the vertebral artery on the same side. In these patients, the blood will flow upward in the opposite vertebral artery, as it normally does, to the vertebrobasilar junction and then will flow downward in the ipsilateral vertebral artery to the subclavian artery beyond the occlusion, restoring the blood supply to that arm. The occlusive lesion is bypassed.[3,8,9,12] In doing so, however, some blood is diverted from the basilar circulation and from the cerebral circulation in general. For this reason, the first manifestation of a subclavian or innominate artery lesion may be a transient cerebral ischemic attack or even a major stroke.[13,26] In some of these patients, the vertebral system is the main collateral trunk to the arm. In others, many arteries participate in establishing a quite intensive collateral circulation, such as: the thyrocervical trunk through the inferior thyroid arteries, the costocervical trunk through the deep ascending cervical arteries, the intercostal arteries and the external carotid arteries.[1,10,16]

In the last 3 years, we have had the opportunity to examine 4 patients in whom retrograde vertebral blood flow was observed on the same side as a subclavian or innominate artery occlusive lesion.

METHOD

The study of the aorta and its brachiocephalic branches in our department is carried out by percutaneous catheterization of either the axillary,[18,20] or femoral artery.[19,21] The injection of opaque medium is done first in the ascending part of the aorta. Roentgenograms include the transverse part of the aortic arch and its branches up to the base of the skull. Fast film changers are used, permitting sequential analysis of the opacified blood flow. After the roentgenograms are inspected, the aortic catheter is passed into the artery or arteries of greatest interest for more detailed study. This technique,[15] we feel, is the most physiologic one, since the opacified blood will flow in the brachiocephalic trunk, the left common carotid and the left subclavian arteries in the same fashion it normally flows.

REPORT OF CASES

CASE 1. W.S., a 41 year old white male, was admitted to the hospital complaining of intermittent episodes of headache, weakness of the legs and dyspnea of 3 years' duration. The attacks consisted of strange sensations in the posterior part of the neck on the right side, radiating to the right frontal region and behind the right eye with concomitant blurring of vision of the right eye. Each episode lasted approximately 2 hours. These attacks occurred every

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second or third day over a period of 1 year. At times, there were as many as 3 per day but during the last 6 months they had been gradually diminishing. In addition, the patient complained of sharp right anterior thoracic pain radiating to the right arm and hand of over 1 year's duration.

Physical examination revealed the right brachial and radial pulses to be reduced and a bruit could be heard over the right sternoclavicular junction, in the right supraclavicular area and at the carotid bifurcation on the same side. The blood pressure in the right arm was 126/100 and in the left arm was 156/106.

While in the hospital, the patient had no attacks but he complained of weakness of his legs on arising in the morning.

Transfemoral arch aortography was carried out. After viewing the roentgenograms, the catheter was inserted into the left vertebral artery and left vertebral angiography was performed. The latter examination showed rapid retrograde filling of the right vertebral artery. In addition, it showed a fairly severe stenosis of the right vertebral artery at its origin. Despite this fact, there was some opacification of the right subclavian artery (Fig. 1, *A* and *B*). The arch aortogram also showed reversal of blood flow in the right vertebral artery thought to be due to narrowing of the right subclavian artery opposite the origin of the right vertebral artery. Attempts were made to obtain better visualization of the innominate artery through selective catheterization but unfortunately this was unsuccessful. There was also a mild degree of stenosis of the right internal carotid artery at its origin (Fig. 2, *A* and *B*).

At operation, atherosclerotic plaques were found at the right vertebrosubclavian junction while exploration of the proximal parts of the

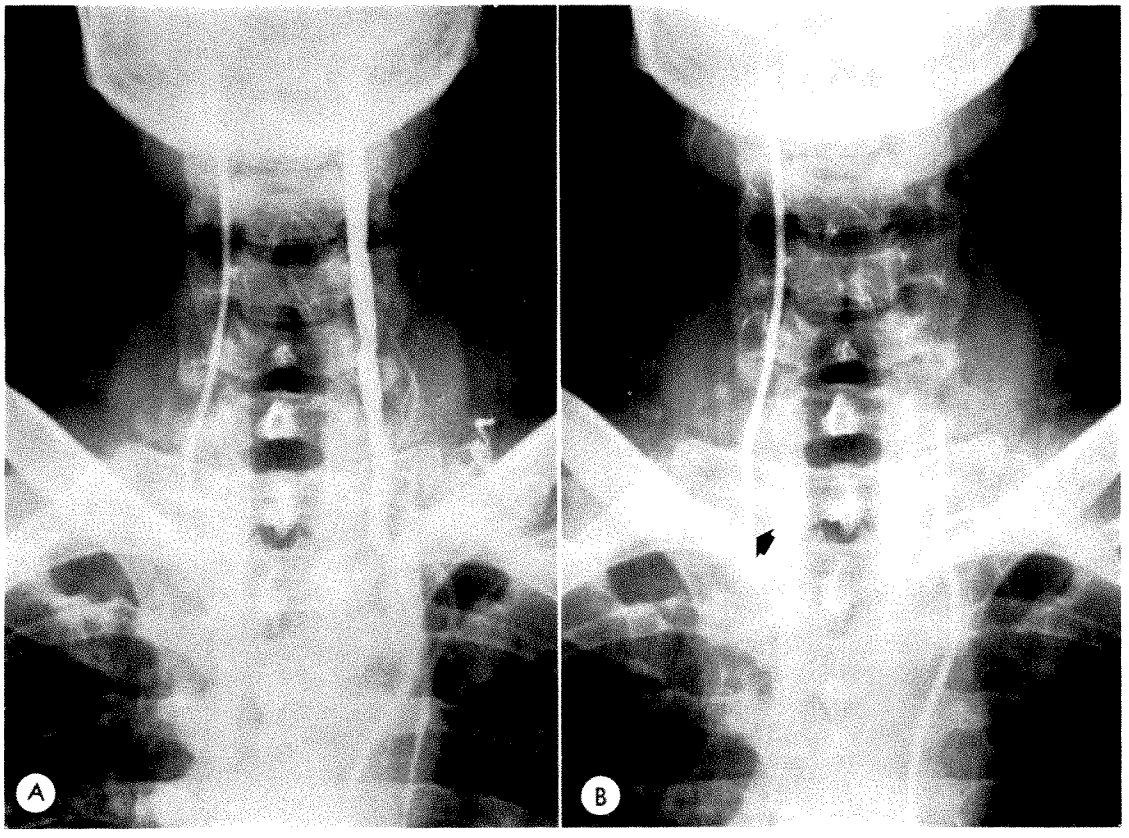


FIG. 1. Case 1. Transfemoral left vertebral angiograms. (*A*) The opaque medium is injected in the left vertebral artery. The immediate roentgenogram shows rapid reversal of blood flow down the right vertebral artery. (*B*) Opacified blood can be seen in the right subclavian artery 1½ seconds later, while more opacified blood is seen in the right vertebral artery. Note the stenotic lesion at the origin of the right vertebral artery (arrow).

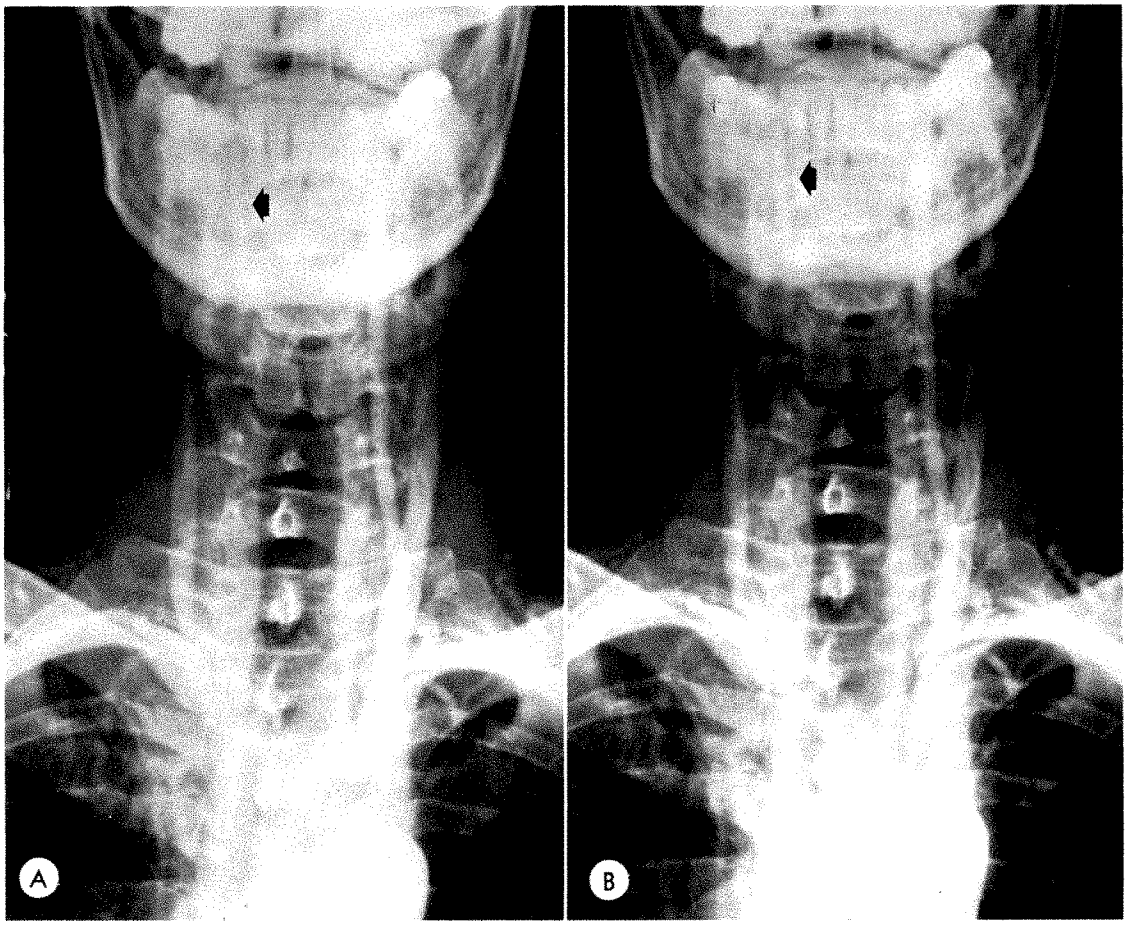


Fig. 2. Case I. Transfemoral arch aortograms. (*A*) Reversal of blood flow is seen in the upper part of the right vertebral artery (arrow). (*B*) The opacified blood in the right vertebral artery can be traced down as far as C7. Minor irregularities of the superior wall of the right subclavian artery opposite the subclavian-vertebral junction and mild stenosis of the proximal segment of the right internal carotid artery secondary to atherosclerosis (arrow) are demonstrated.

subclavian as well as of the innominate artery revealed no gross lesion. On the second postoperative day, the patient developed occlusion of the right subclavian artery. The pulse in the right arm became extremely weak and no blood pressure could be recorded. However, no signs of cerebral insufficiency or brachial insufficiency developed; therefore, no further operation was performed. Anticoagulant therapy was instituted. A year after operation, the patient is still under anticoagulant therapy and has no neurologic complaints.

Case II. B.C., a 51 year old white male, was admitted to the hospital with a history of a severe attack of dizziness, vertigo, and vomiting, lasting for about 24 hours. This acute episode happened while the patient was chopping wood, using his right arm. After the acute episode, the patient remained free of any neurologic symptoms for 1 year when he had a sudden onset of focal motor seizures, involving the left arm and face followed by weakness of the left hand.

Physical examination revealed weak right radial and brachial pulses. The blood pressure in the right arm was 108/70, while it was 130/80 in the left arm. Mild hyper-reflexia in the left arm and leg was noted, as well as a cortical type of sensory loss over the left side of the body.

Transfemoral arch aortography was carried out and revealed complete occlusion of the proximal segment of the right subclavian artery. Opacified blood was seen passing down the

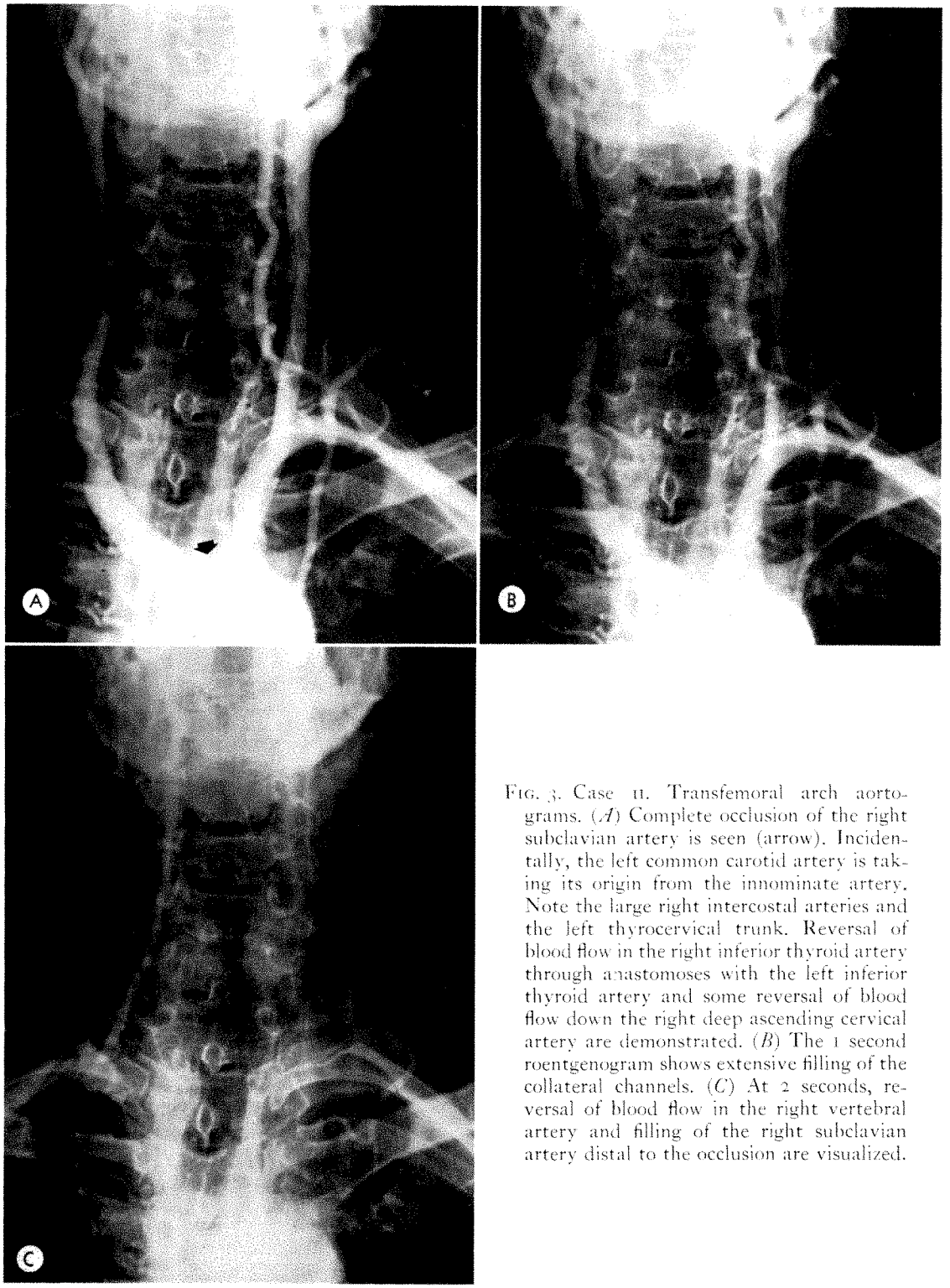


FIG. 3. Case 11. Transfemoral arch aortograms. (*A*) Complete occlusion of the right subclavian artery is seen (arrow). Incidentally, the left common carotid artery is taking its origin from the innominate artery. Note the large right intercostal arteries and the left thyrocervical trunk. Reversal of blood flow in the right inferior thyroid artery through anastomoses with the left inferior thyroid artery and some reversal of blood flow down the right deep ascending cervical artery are demonstrated. (*B*) The 1 second roentgenogram shows extensive filling of the collateral channels. (*C*) At 2 seconds, reversal of blood flow in the right vertebral artery and filling of the right subclavian artery distal to the occlusion are visualized.

right vertebral artery and the right subclavian artery distal to the lesion on the late roentgenograms. In addition, extensive collateral circulation other than the vertebral circulation had appeared. The right intercostal arteries were much larger than the left. There was reversal of blood flow in the deep ascending cervical artery, as well as in the branches of the thyrocervical trunk on the right side. These latter vessels were seen to fill from the corresponding branch on the left side, namely, the inferior thyroid artery (Fig. 3, *A*, *B* and *C*).

An incidental finding was the left common carotid artery taking origin from the innominate artery rather than from the aortic arch itself.

Following this, selective catheterization of the right common carotid artery was performed and the right carotid angiogram showed an avascular area, most likely a cerebral infarct in the right parietal lobe. Good filling of the right posterior cerebral artery from the internal carotid artery was obtained suggesting active participation of the carotid system in the posterior part of the circle of Willis. The occipital artery on the right side was well developed and showed multiple anastomoses with the deep ascending cervical artery (Fig. 4).

Although surgical treatment was considered, it was not applied. The patient has remained apparently asymptomatic since his admission almost a year ago, except for a second seizure involving the left side of his body.

Case III. R.I., a 55 year old white female, was admitted to the Montreal Neurological Institute after sudden onset of paralysis of the left arm which progressed to involve the whole left side of the body. This was associated with a sudden blurring of vision of the right eye, which lasted approximately 1 minute. There was no pulse in the right arm during this acute episode and no blood pressure could be recorded. Shortly after her admission, the blood pressure in the right arm was 80/60 and it was 130/80 in the left arm. A bruit could be heard over the right supraclavicular area as well as over the bifurcation of the left carotid artery. Before the acute episode, the patient had had attacks of blanching of the fingers of the right hand which were unrelated to cold. This was associated with a dull ache in the right elbow. Normal blood pressure in the right arm was said to be present then.

Transfemoral arch aortography was carried out and showed complete occlusion of the innominate artery extending up to the first segment of the right subclavian artery. Consequently, the right common carotid artery was not visualized. Opacified blood was seen to flow down the right vertebral artery. Subsequently, there was moderately good opacification of the right subclavian artery. There was, in addition, some suggestion of retrograde filling of the deep ascending cervical artery on the right side probably through anastomosis between the external carotid arteries (Fig. 5, *A*, *B* and *C*).

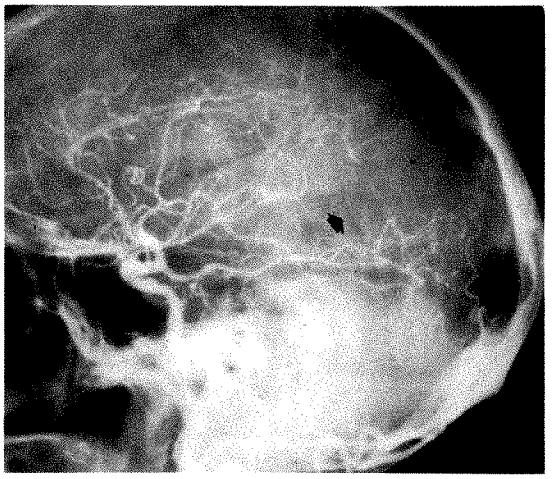


Fig. 4. Case II. Transfemoral right carotid angiogram showing an avascular area in the right parietal lobe (arrows), thought to be an infarct. Note the active participation of the carotid system in the posterior part of the circle of Willis. Well developed anastomoses between the occipital branches of the external carotid artery and the deep ascending cervical artery in which the blood is seen to flow in a retrograde fashion are demonstrated.

During the procedure, the patient developed cardiac arrest which was treated by extrathoracic massage and the external defibrillator. For this reason, no surgical treatment was attempted.

Case IV. L.A., a 43 year old white male, had a 7 year history of periodic attacks of sudden vertigo lasting for approximately 10 minutes. These attacks, which occurred every 6 or 7 months, were associated with a feeling of weakness and dizziness as well as perspiration and nausea. Shortly before admission, he had a similar acute episode with loss of consciousness.

He had ataxia of the left hand and left leg, as well as sensory disturbances of the left leg,

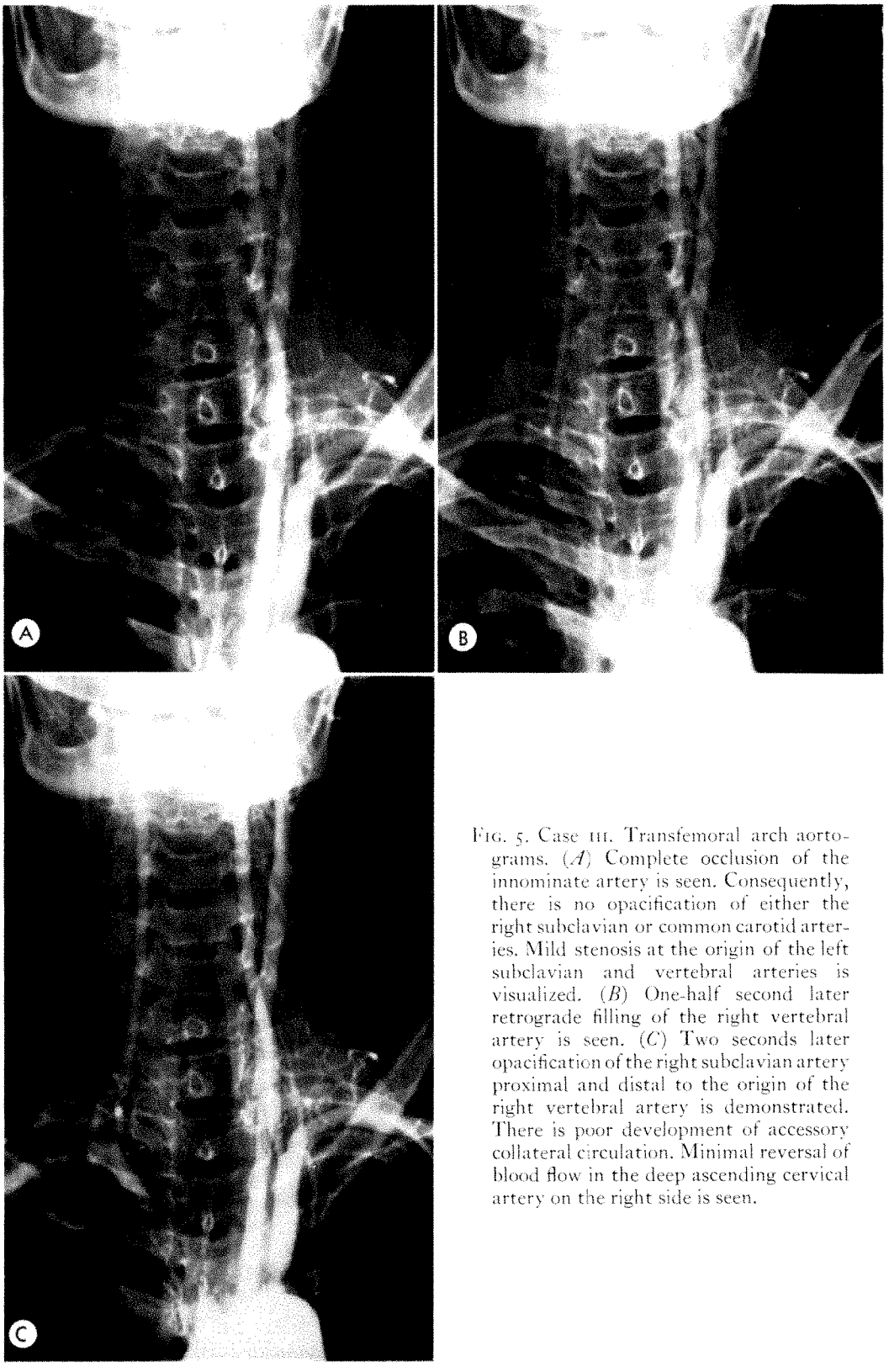


FIG. 5. Case III. Transfemoral arch aortograms. (*A*) Complete occlusion of the innominate artery is seen. Consequently, there is no opacification of either the right subclavian or common carotid arteries. Mild stenosis at the origin of the left subclavian and vertebral arteries is visualized. (*B*) One-half second later retrograde filling of the right vertebral artery is seen. (*C*) Two seconds later opacification of the right subclavian artery proximal and distal to the origin of the right vertebral artery is demonstrated. There is poor development of accessory collateral circulation. Minimal reversal of blood flow in the deep ascending cervical artery on the right side is seen.

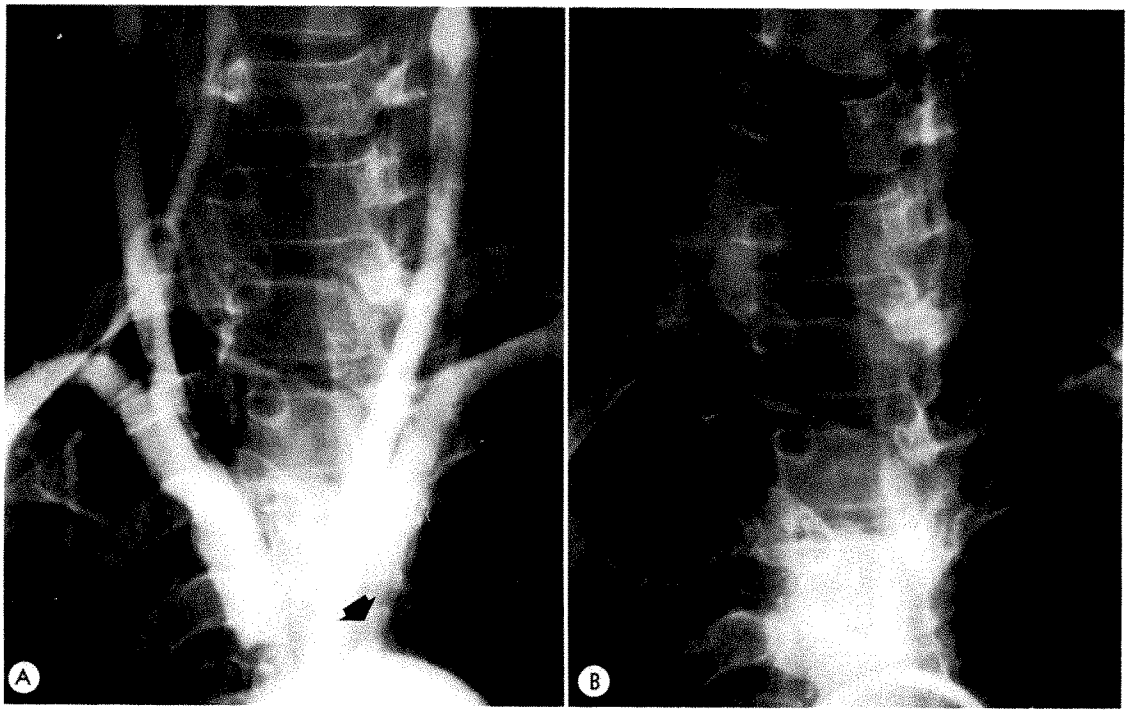


Fig. 6. Case IV. Transfemoral arch aortograms. (*A*) Stenotic lesions are seen at the origin of the innominate artery (arrow), both subclavian arteries (arrows) and both internal carotid arteries. There is no opacification of the left vertebral artery. (*B*) Reversal of blood flow in the left vertebral artery and reopacification of the left subclavian artery distal to the origin of the vertebral artery are demonstrated. No collateral circulation other than the vertebrobasilar system is visible.

hand, tongue and face and dysarthria. The blood pressure was 116/80 in the right arm and 85/65 in the left arm.

A transfemoral arch aortogram showed stenotic lesions involving the origin of the innominate artery as well as the origin of both subclavian arteries and of both internal carotid arteries. The left vertebral artery was seen to fill from above downwards, contributing to the blood supply of the left arm as shown by the faint opacification of the left subclavian artery (Fig. 6, *A* and *B*). In this patient, no collateral circulation other than the vertebrobasilar system was visible.

DISCUSSION

Atherosclerosis is a multifocal disease and it is difficult to predict in any one patient its extent.[27] The clinical picture will vary remarkably from one patient to another depending upon the multiplicity of the lesions, the severity of the lesions, and the development of collateral circulation.[7,25] Therefore, it is difficult to predict on clinical grounds which of the aortic vessels is responsible for the clinical picture in cerebrovascular disease. For this reason, it is essential to visualize the aortic arch and the whole length of its branches by an injection into the arch itself. Anomalies of the aortic arch will be demonstrated readily which otherwise can be missed and therefore misinterpreted. The relative rate of blood flow in the neck vessels can be appreciated in terms of relative speed and relative density of opacified blood.

Reivich and co-authors[17] have clearly demonstrated the effects of reversal of blood flow in the vertebral arteries associated with stenosis or obstruction of the subclavian arteries. According to their canine experiments, obstruction of the left subclavian artery was diverting up to 41 per cent of the total cerebral blood flow. However, in cases where partial rather than

complete occlusion of the subclavian arteries is present, the reduction in cerebral blood flow must be less. Furthermore, in patients where extensive collateral circulation has developed (such as in Case II) so that the blood supply to the arm comes only partially from the vertebral system, the detracting effects on the cerebral circulation are less pronounced. Contrasting with the cerebral ischemia which might be quite severe in those patients, the ischemia in the affected arm seems to be relatively mild, although the blood pressure difference between the two arms is sometimes quite marked. An interesting feature is that the cerebral ischemic attack can be precipitated by exercise of the affected arm as in the second case of our present series.

More cases of this condition are beginning to appear in the medical literature.[24] This is probably due to the fact that blood pressure and radial pulses are now recorded in both arms in patients showing signs of cerebral ischemia. This should become a standard procedure in every patient presenting cerebrovascular manifestations.

Early recognition of this syndrome, called by some investigators the "subclavian steal" and by others the "brachial-basilar insufficiency syndrome," before permanent cerebral damage occurs is of the utmost importance.[6,14] If surgical corrective measures are applied early, they are most likely to be beneficial.[11]

SUMMARY

Occlusive disease, partial or complete, involving the proximal segment of the subclavian arteries or innominate artery, is counteracted by the development of a collateral circulation in which retrograde vertebral artery blood flow plays a major role. If the vertebral arteries alone are responsible for maintaining an adequate blood supply to the arm on the side of the innominate or subclavian lesion, the effects on the cerebral circulation may be highly detrimental. These effects will vary from patient to patient depending upon the degree of stenosis of the brachiocephalic vessels and the general condition of the other neck vessels. The development of an extensive collateral circulation other than the vertebral system is apt to reduce the diversion of blood from the cerebral circulation.

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ADDENDUM

Since the completion of this article, we have had the opportunity to carry out transaxillary aortography in a patient who had a sudden onset of hemiplegia on the left side associated with decreased blood pressure and pulse volume in the left arm.

This examination and right carotid angiography done immediately after with the same catheter showed that most of the blood to the brain was carried by the right carotid system. There were severe stenotic lesions involving both subclavian arteries at their origin as well as the left common carotid artery at its origin. Reversal of blood flow was present in the left vertebral artery as well as in the basilar artery. In addition, on successive roentgenograms blood was seen to flow in both directions, in the upper part of the right vertebral artery and in the subarachnoid segment of the left internal carotid artery.

It seems that many aspects of the dynamics of blood flow in the neck vessels remain to be appreciated. From a roentgenologic standpoint, the combined use of catheterization of the aortic arch and cineangiography will probably help to determine some of the practical aspects.[23]

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SUBCLAVIAN STEAL SYNDROME

PITFALLS IN ITS DIAGNOSIS

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THE astute observations and investigations of Reivich and his co-workers[3] in 1961 clearly established the presence of a new syndrome to which the term "subclavian steal" was applied by medical editorialists in the *New England Journal of Medicine*. The recognition of this new entity has aroused widespread interest and many new cases are now being reported in current medical literature.[1,2,5] This may prove to be one of the most important causes of cerebral arterial insufficiency amenable to surgical relief.

It is the purpose of this communication to indicate that overzealous efforts to identify this newly recognized entity may have resulted in the reporting of some cases which are not true subclavian steals. Recent experiences convince us that this diagnosis can be made erroneously. In our opinion, this is particularly true when the entity is established by roentgenologic studies limited to a unilateral retrograde brachial arterial study.

It is our belief that the diagnosis is particularly subject to error unless arch aortography or contralateral retrograde brachial arteriography is performed. The following case briefly illustrates this point.

REPORT OF A CASE

CASE I. A 71 year old white female was hospitalized 1 week after onset of coma. Right retrograde brachial arteriography was performed (Fig. 1, *A*, *B* and *C*). Routine brachiocephalic studies showed delayed filling of the left vertebral and subclavian arteries, but at the same time considerable residual opaque medium in the distal aortic arch. Occlusive narrowing of the left subclavian artery was also suspected on delayed roentgenograms, and a diagnosis of "subclavian steal" was considered. Since the proximal left subclavian artery was incompletely visualized, it was difficult to determine whether the left vertebral filling was antegrade or retrograde. Serial ½ second roentgenograms in this case did not clarify the problem.

The patient expired 4 days later, and postmortem arch aortography was performed using a simplification of the method described by Stein and Svare.[4] The aortic arch was divided at its origin and ligated just distal to the left subclavian artery. Following gentle irrigation with saline, the aortic stump was closed and an aqueous mixture of sodium iodide and corn syrup was injected until the arch and brachiocephalic trunks were slightly distended. Figure 1*D* shows antegrade filling of the left vertebral artery with only moderate sclerotic narrowing of the proximal left subclavian artery.

Roentgenograms are shown of 2 other cases (Fig. 2, *A* and *B;* and 3, *A* and *B;* Case II and III) simply to emphasize the value of arch aortography in the thorough evaluation of the subclavian steal syndrome. In Case II, symptoms of right brachial claudication and syncope were promptly relieved following endarterectomy. In Case III, the patient's age and extremely poor clinical status precluded any likelihood of relief from surgery. We have found only 2 other instances of common carotid occlusion reported to be associated with the subclavian steal syndrome; but since many of the reports are based on unilateral retrograde brachial studies, the true incidence is not known.

SUMMARY AND CONCLUSIONS

1. Many recent case reports in the literature establish a diagnosis of "subclavian steal" based on what is apparently unilateral retrograde brachial arteriography. Under such circumstances, serial roentgenograms do not always indicate whether delayed contralateral vertebral filling is antegrade or retrograde.

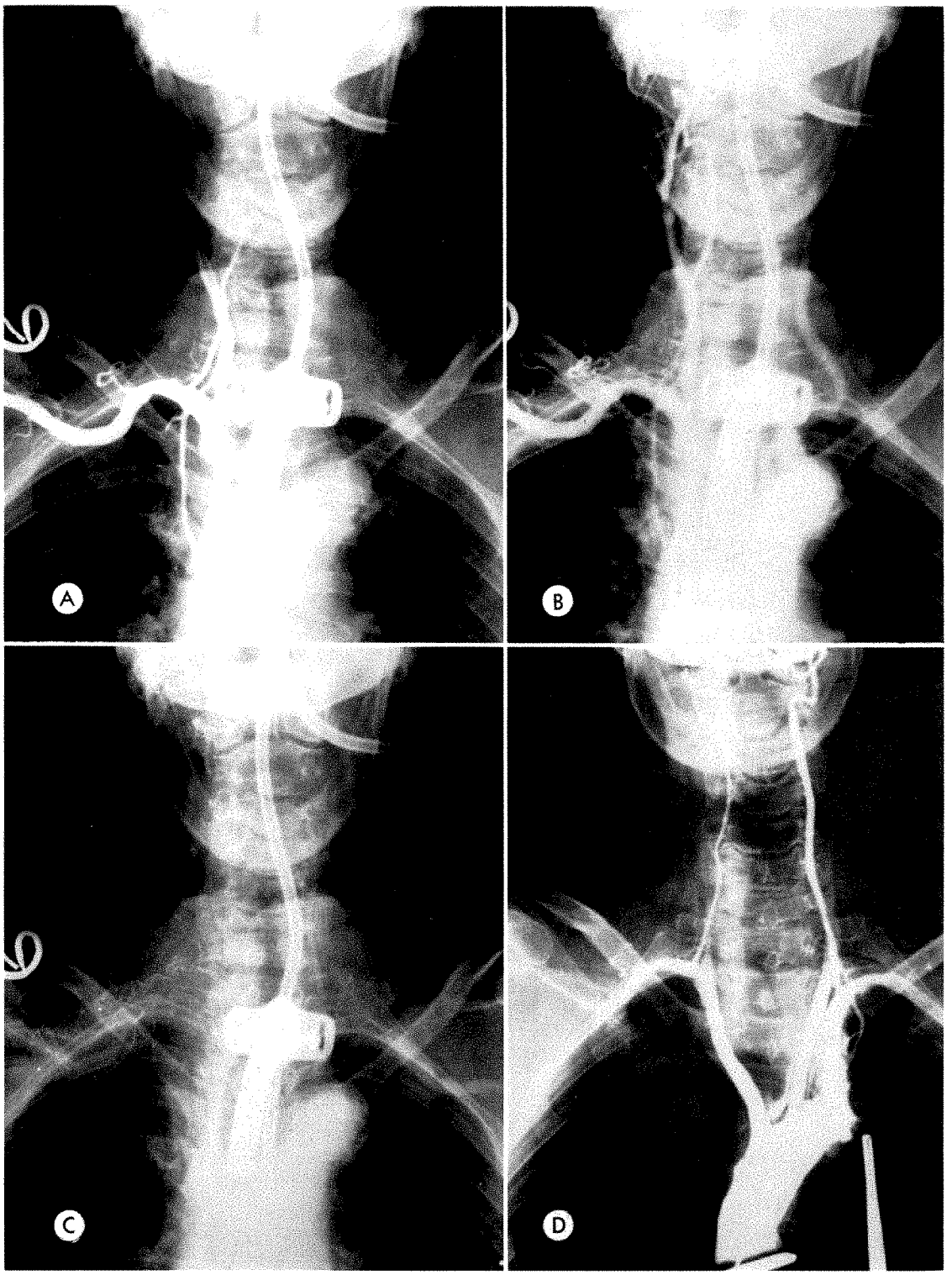


FIG. 1. Case 1. Erroneously diagnosed left subclavian steal. Right brachial arteriography was performed for evaluation of a 71 year old comatose female. Blood pressure in right arm 140/70; in left arm 140/70. (*A*) One second after injection of 30 ml. of 50 per cent diatrizoate sodium (hypaque) into cannulated right brachial artery with pressure of 525 lb. sq. in. ($\frac{1}{2}$ second interval roentgenograms). Early normal fill of right brachiocephalic arteries is shown with a small "puff" of opaque material entering the aortic arch. (*B*) One second after *A*, the distal aortic arch is well filled showing all major brachiocephalic arterial trunks except the distal left subclavian and vertebral. Only the proximal left subclavian artery is filled with evidence suggesting a stenotic occlusion just above the aortic arch. (*C*) One second after *B*, showing delayed visualization of the left subclavian and vertebral arteries with persistent considerable opaque medium in the aorta. (*D*) Postmortem arch aortogram clearly establishing patency of a sclerotic left innominate artery with free antegrade filling of the left vertebral artery.

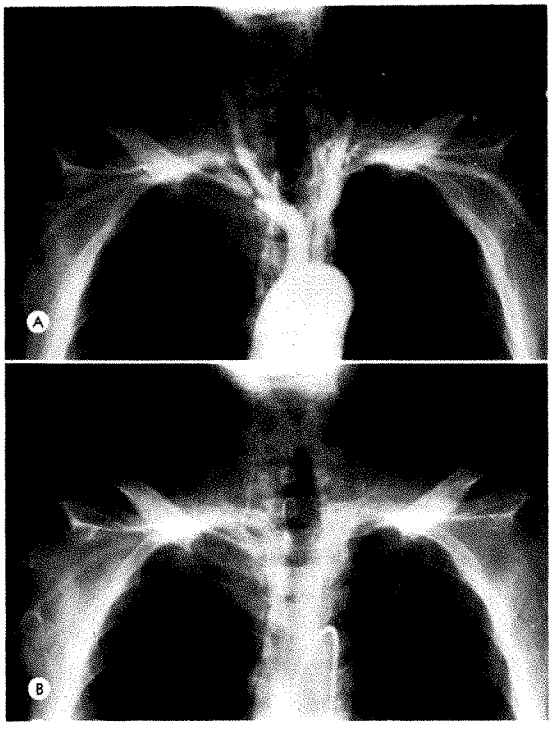


FIG. 2. Case II. Right-sided subclavian steal. Arch aortography performed for evaluation of right arm claudication associated with vertigo and syncope (51 year old male). Blood pressure in right arm 100/70; in left arm 155/105. (*A*) One-half second after injection of 30 ml. of sodium iothalamate 80 per cent (angio-conray) through catheter introduced in the femoral artery percutaneously, Cordis pressure injector—500 lb./sq. in. A sharply defined incomplete occlusion of the proximal right subclavian artery is shown. (*B*) Roentgenogram made 2½ seconds after *A* showing delayed visualization of the right vertebral artery. Intervening serial roentgenograms supported left retrograde vertebral flow.

2. Our observations, recorded in this communication, convince us that a diagnosis of "subclavian steal" may easily be

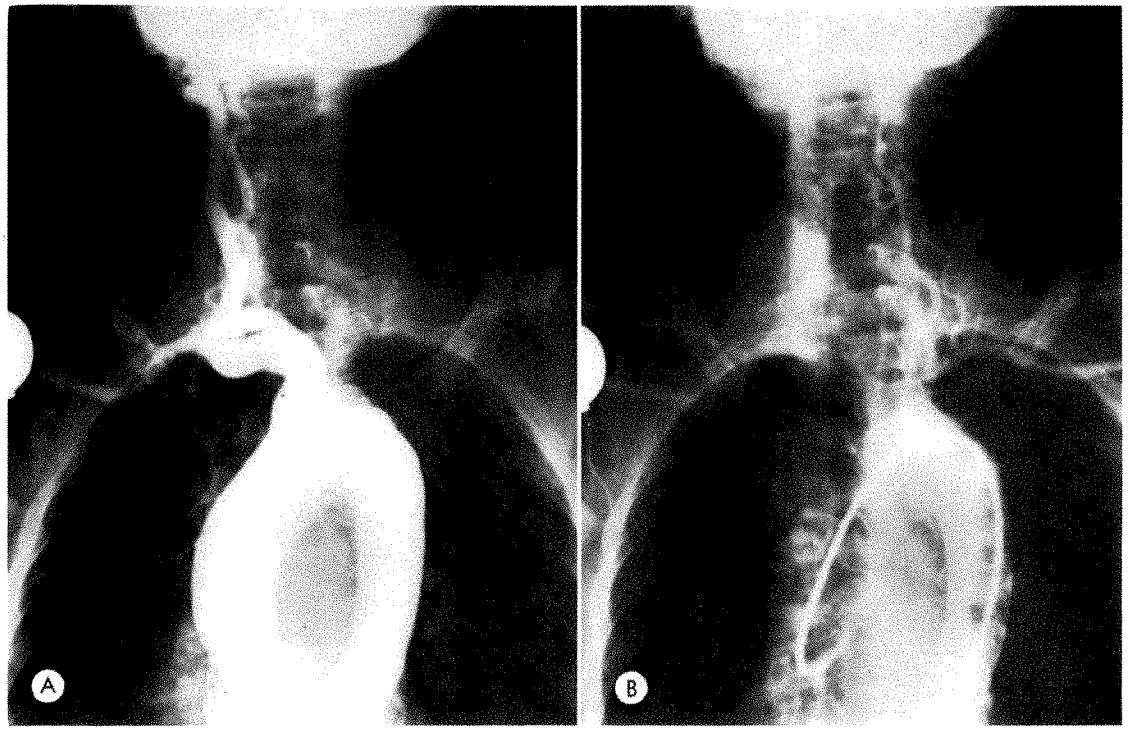


FIG. 3. Case III. Left-sided subclavian steal. Arch aortography was performed for evaluation of a pulsatile right neck mass which was shown to be due to a tortuous innominate artery. There was left hemiplegia of 12 years' duration (71 year old female). Blood pressure in right arm 170/100; in left arm 90/60. (*A*) One-

(*Legend continued on next page*)

made erroneously if the roentgenologic examination excludes arch aortography or bilateral retrograde arteriography. Arch aortography seems to us to be the most simple and physiologic method of detailed evaluation of brachiocephalic vessels.

3. Unilateral right retrograde brachial arteriography commonly fails to clearly establish the detailed lumen status of the opposite proximal subclavian artery.

4. Finally, we are convinced that both the vascular surgeon and the patient are entitled to maximum detailed information prior to surgery for the relief of arterial occlusive disease.

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half second after injection of 35 cc. of sodium iothalamate 80 per cent (angio-conray) through a catheter introduced in the femoral artery percutaneously. There is definite evidence of occlusion of the left subclavian and left innominate arteries at their aortic arch origins. (*B*) Roentgenogram made 2½ seconds after *A*, showing delayed filling of the left vertebral and distal subclavian arteries. Intervening serial roentgenograms supported left retrograde vertebral flow.

RETROGRADE VERTEBRAL ARTERY FLOW AS AN ARTIFACT OF TECHNIQUE*

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AN INTERESTING neurologic syndrome caused by obstruction of the subclavian artery proximal to the origin of the vertebral artery has recently been described and has attracted widespread attention.[2,3,5–8,10,11] Blood to the involved arm is supplied primarily by retrograde flow from the ipsilateral vertebral artery into the subclavian artery distal to the site of obstruction. This "subclavian steal" syndrome is characterized clinically by diminished blood pressure in the extremity and by symptoms of cerebral ischemia. The neurologic symptoms are frequently aggravated by strenuous use of the arm which results in increased blood flow to the upper extremity at the expense of the brain. A bruit can sometimes be heard in the region of the subclavian narrowing. Roentgenologically, the syndrome is characterized by the demonstration of narrowing of the subclavian artery near its origin from the aorta or innominate artery and by visualization of retrograde flow of contrast material down the vertebral artery on the side of the obstruction. A few cases have been reported in which the diagnosis was based primarily on angiographic findings either in the absence of cerebral ischemic symptoms or in the presence of symptoms that could readily have been related to atherosclerotic disease demonstrated elsewhere.[2,3,6,7,8]

We have recently encountered 2 patients with symptoms of cerebral ischemia in whom arteriographic studies revealed retrograde flow in a vertebral artery suggesting the presence of the "subclavian steal" syndrome. In both cases, however, further studies showed that the retrograde vertebral flow of contrast material was probably an artifact.

REPORT OF CASES

CASE 1. A 62 year old white man was hospitalized because of syncopal episodes and weakness in his legs after walking short distances. The pulsation of the left carotid artery was weaker than that of the right. Neurologic examination revealed no abnormalities. The blood pressure was 114/60 in the right arm and 106/60 in the left arm. Right retrograde brachial arteriography was performed through a catheter whose tip was located at the junction of the right subclavian and common carotid arteries. Twenty cubic centimeters of 85 per cent cardiografin were injected at 9 kg./cm.^{2} pressure (Gidlund). The arteriogram showed filling of the right vertebral artery and delayed retrograde filling of the left vertebral artery (Fig. 1, *A* and *B*), subclavian artery and aorta. After a slight adjustment of the catheter, a second injection using 20 cc. of 60 per cent renografin showed filling of the right vertebral and common carotid arteries but no evidence of retrograde filling of the left vertebral artery. Percutaneous right carotid arteriography was normal. A few days later an intravenous aortogram utilizing 90 cc. of 85 per cent cardiografin showed that all four major neck vessels were normal. A second injection demonstrated a patent basilar artery.

Comment. Retrograde filling of the left vertebral artery was seen on one occasion but the entire left subclavian artery from its aortic origin to the axillary artery was visualized and seen to be free of obstructive lesions. The reversal of flow in the left vertebral artery was not seen following a second injection shortly thereafter. The only changes between the two injections

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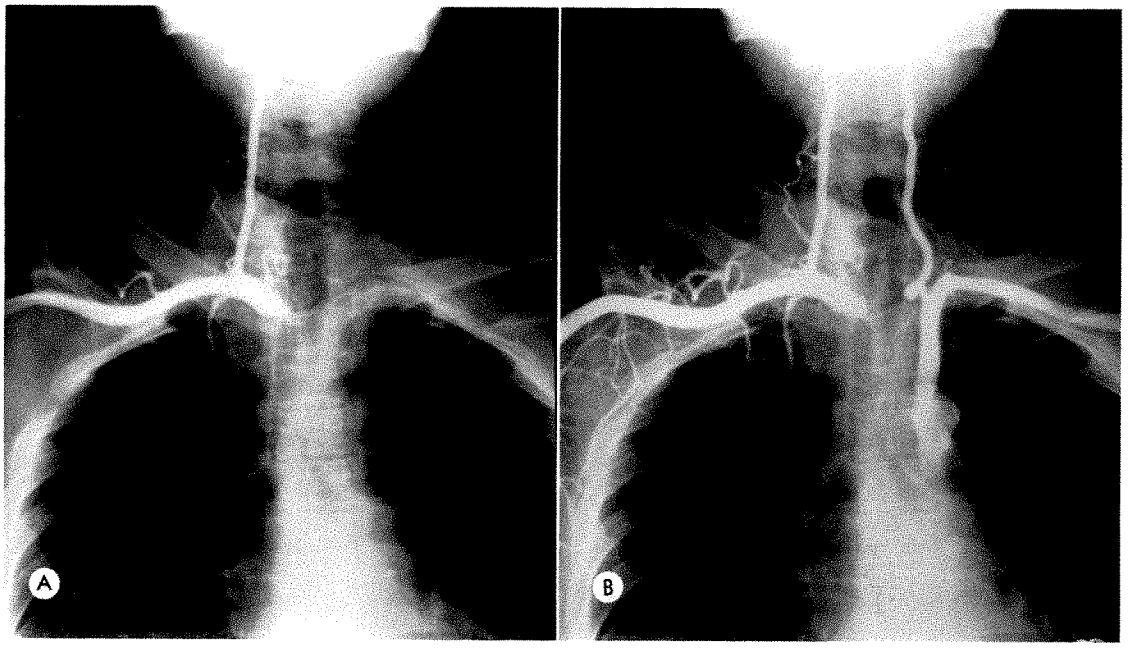


Fig. 1. Case I. (*A*) Right retrograde brachial arteriogram demonstrating right vertebral artery. (*B*) Later study demonstrating reflux down left vertebral artery filling entire left subclavian artery from aorta to axillary artery.

were the position of the catheter tip and the contrast agent used.

Case II. A 69 year old Negro with a 10 year history of hypertension was hospitalized because of weakness and tingling in the right forearm of 6 days' duration. For 48 hours prior to hospitalization he had noted difficulty with speech and mild frontal headache. The left carotid pulse was weak relative to the right. The blood pressure was 150/105 in both arms. On neurologic examination minimal weakness of the right forearm with a slight increase in the tendon reflexes was noted. No pathologic reflexes were found. All peripheral pulses were palpable. All laboratory examinations were within normal limits except for a positive serologic test for syphilis. Left brachial arteriography was performed through a catheter whose tip was located in the subclavian artery at the origin of the vertebral artery. Thirty cubic centimeters of 85 per cent cardiografin were injected at 9 kg./cm.^{2} pressure (Gidlund). The left vertebral artery was seen followed by delayed retrograde filling of the right vertebral artery (Fig. 2, *A* and *B*). A few days later a catheter was placed in the right brachial artery and injection of contrast material showed normal right subclavian and right vertebral arteries (Fig. 3).

Comment. Again retrograde filling of a vertebral artery was seen although a later study revealed the subclavian artery on the side of flow reversal to be without obstruction.

DISCUSSION

Surgical correction of the underlying obstructive lesion has been followed by complete relief of the "subclavian steal" syndrome in a few instances. One may, therefore, confidently recommend surgery when there is clinical evidence of diminished arterial perfusion of an upper extremity associated with cerebral ischemic manifestations aggravated by use of the involved extremity and by roentgenologic evidence of obstruction in the subclavian artery at or near its origin with retrograde flow of contrast material down the ipsilateral vertebral artery. On the other hand, when the syndrome is incomplete, one must be prognostically cautious since atherosclerosis

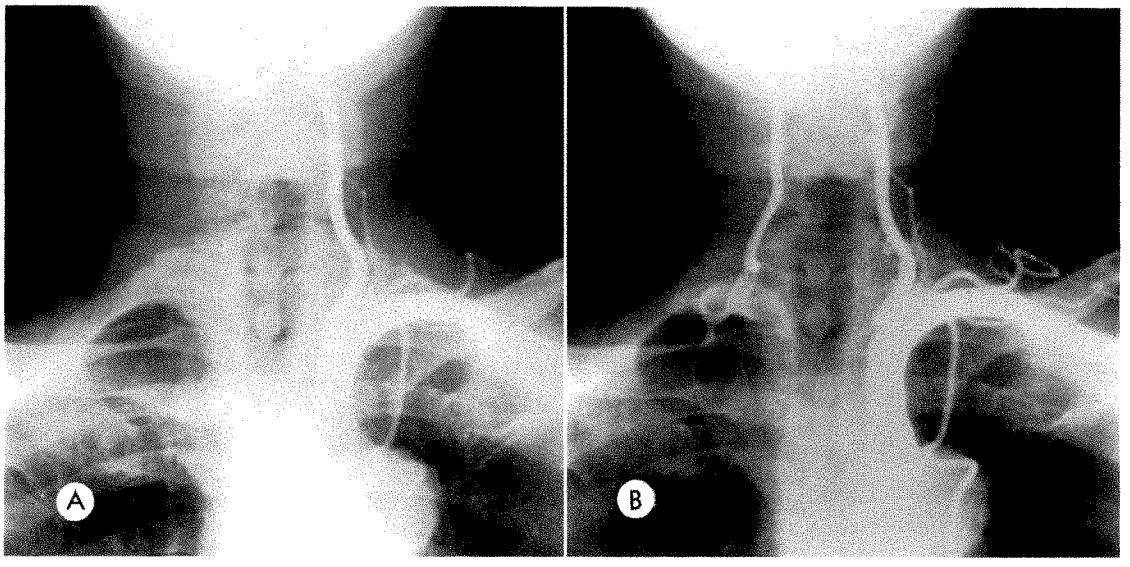


Fig. 2. Case II. (*A*) Left retrograde brachial arteriogram demonstrating left vertebral artery. (*B*) Later study showing retrograde flow down right vertebral artery.

tends to be a widespread disease and lesions elsewhere in the cerebral circulation may well account for the ischemic symptoms. The 2 cases reported in this article demonstrate that one must be particularly cautious in interpreting the significance of the arteriographic demonstration of retrograde flow of contrast material in the vertebral arteries. Although this is a striking roentgen finding, our 2 patients illustrate the fact that it may be artifactual since there was no evidence of obstruction in the subclavian artery proximal to the origin of the vertebral artery and other arteriographic studies showed normal vessels with no evidence of retrograde flow.

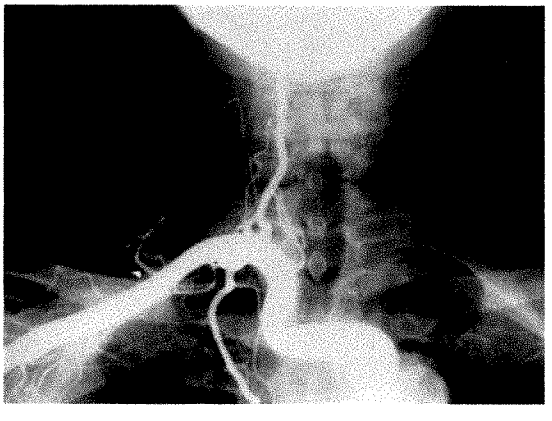


Fig. 3. Case II. Right retrograde brachial arteriogram demonstrating absence of obstruction in right subclavian artery, anomalous origin of right subclavian artery and absence of retrograde flow in left vertebral artery.

In both cases there was difficulty in advancing the tip of the catheter beyond the point at which injection was made. Conceivably, because of this, the end hole was blocked by the vessel wall and the greatest part of the high pressure exerted and contrast material injected was delivered via a side hole directly into the vertebral artery. The diminished elasticity of older arteries also may have facilitated the transmission of pressure relatively undiminished into the vertebrobasilar region producing momentary flow reversal.

It has been demonstrated by Amundsen *et al.*[1] that a marked significant fall in blood pressure (30–40 mm. Hg) occurs with aortography, peripheral arteriography, and angiocardiography. Rowe *et al.*[9] also described a precipitous drop in peripheral arterial pressure in man and confirmed this experimentally in dogs following angiocardiography. Lindgren and Törnell[3] attributed the fall in blood pressure to a vasodilator effect of the contrast agents.

They also established that there was a disproportionately greater effect with more concentrated preparations of the same agents. It seems possible, therefore, that a general fall in blood pressure, locally manifest in the subclavian artery, occurred at a time when the pressure at the vertebrobasilar junction was at a peak, thereby creating a transient reversal of the pressure gradient in the vertebral artery.

The roentgen evaluation of cerebrovascular disease is hazardous when based on arteriographic studies which show only a portion of the circulation to the neck and head. Several authors[7,8,10] have emphasized that all the major branches from the aorta to the intracranial level should be visualized simultaneously with rapid serial roentgenography in order to obtain information regarding the hemodynamics in the individual patient. The demonstration of a localized stenosis or obstruction is not adequate for planning the over-all treatment program. The direction of flow in an artery can be transiently altered by counter-current arterial injections, thus masking retrograde flow on the ipsilateral side[10] as well as artifactually producing it on the contralateral side. The technique which most clearly reveals the true direction of flow in the major arteries is intravenous injection with delayed serial roentgenography of the aortic arch and its branches. Since this procedure does not always produce adequate contrast, injection through a catheter in the arch of the aorta may be preferred. If counter-current brachial or subclavian artery injection is used, a noncatheter technique is desirable. If intra-arterial catheter techniques are to be reliably utilized, care must be exercised that the tip of the catheter is located well away from the vertebral orifice, that the less concentrated radiopaque media are used and that the injection pressure is kept as low as is compatible with good visualization. If reversal of vertebral artery flow is demonstrated, its presence should probably be confirmed by use of an alternate technique. In addition, definite anatomic obstruction in the subclavian artery proximal to the vertebral artery must be demonstrated.

SUMMARY AND CONCLUSIONS

1. Two cases are presented in which transient retrograde blood flow was noted angiographically despite the absence of obstruction in the ipsilateral subclavian artery.

2. The artifactual reversal of blood flow is ascribed to the technique employed.

3. Technical recommendations for averting this artifact are offered.

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VOL. 91, NO. 6

DISSECTING ANEURYSMS OF THE THORACIC AORTA*

THEIR ANGIOGRAPHIC DEMONSTRATION

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A DISSECTING aneurysm of the thoracic aorta is an intramural hematoma which commonly begins as a transverse tear in the intima and media of the aorta a few centimeters above the aortic valve or in the descending thoracic aorta near the origin of the left subclavian artery.[3] It differs from other aneurysms in that the aortic lumen is usually not enlarged by the dissection, but remains constant in size or is made smaller by the hematoma.[10,12]

The recent advances in cardiovascular surgery and the general employment of vascular opacification have made it essential to appreciate the roentgen findings of dissecting aneurysm of the thoracic aorta. The following 15 cases studied during the last 24 months confirm the frequency of this pathologic process.

The increasing age of the general population, the large number of hypertensives in western society and the relationship of medial arterial necrosis to Marfan's syndrome, a heritable disorder of the connective tissue, are indicative of the ever increasing number of cases that will be encountered in the future.

REPORT OF CASES

CASE I. This 52 year old white female was admitted to the hospital for a cholecystectomy for cholelithiasis. No symptomatology was referable to the chest.

Physical Examination. On admission the patient's blood pressure was 110/70, pulse 88, and respiration 18. No murmurs were heard upon examination of the chest.

Roentgenologic Findings. Indirect: Examination of the chest showed a dilated aortic arch which appeared to have increased in size when compared to a previous roentgenogram. In addition, there was a definite suggestion of a double contour to the aortic knob. It was felt that the plain roentgenogram suggested a dissecting aneurysm of the thoracic aorta (Fig. 1 and 2). Direct: An intravenous aortogram demonstrated the dissection of the thoracic aorta. Dissection began at the ascending portion of the arch and involved the origin of the main thoracic branches and extended along almost the entire thoracic aorta. The point of re-entry was thought to be in the lower thoracic aorta just at the level of the diaphragm. The opaque material was seen to penetrate the dissected portion of the aorta (Fig. 3 and 4).

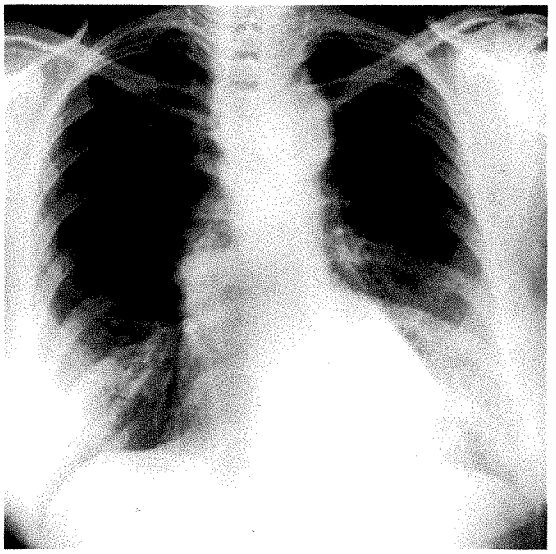


FIG. 1. Case 1. Frontal chest roentgenogram made 1 year prior to admission reveals some left ventricular prominence and unfolding of the aortic arch. The lung fields are unremarkable.

CASE II. This patient is a 55 year old female who entered the hospital with the chief complaint of soreness in the midepigastrium of 5 days' duration. The pain did not radiate.

Physical Examination. Physical examination

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Presented at the Sixty-fourth Annual Meeting of the American Roentgen Ray Society, Montreal, Quebec, Canada, October 8–11, 1963.

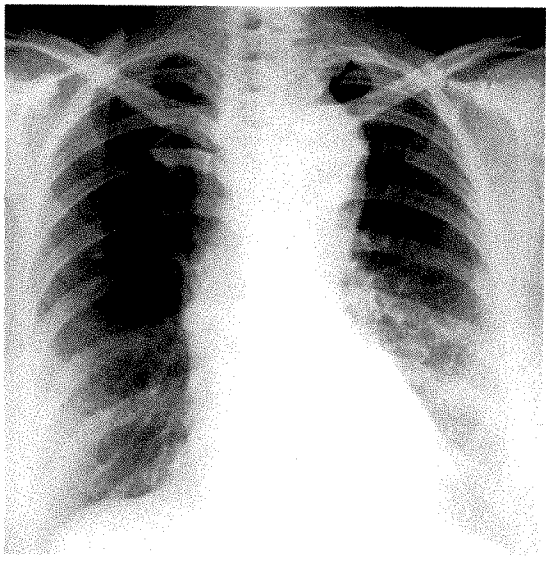


FIG. 2. Case 1. Chest roentgenogram made during the present admission shows definite increase in the dilatation of the aorta, most striking in the descending portion of the arch and the thoracic aorta. The contour of the aortic knob as outlined by the black arrows shows a definite irregularity as well.

revealed a loud aortic systolic murmur and an apical systolic murmur. The clinical impression was that of a possible abdominal aortic aneurysm.

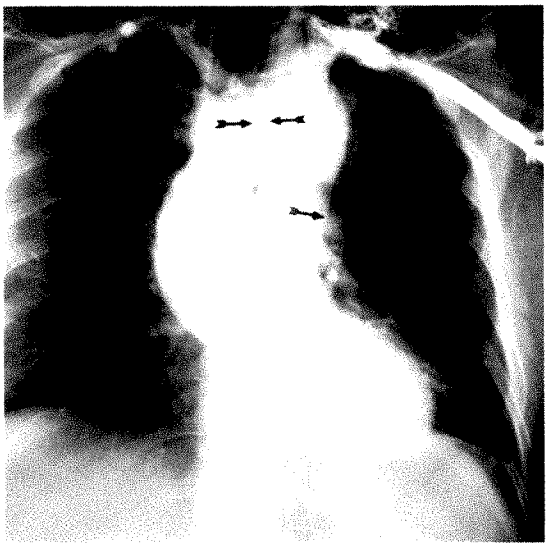


FIG. 3. Case 1. Frontal angiocardiogram shows two well defined false channels of contrast material (top arrows). The thickness of the wall of the descending aorta is secondary to the dissection. The lower arrow delineates the lateral wall of the true aortic lumen.

Roentgenologic Findings. Indirect: Chest examination revealed an enlarged heart with dilatation of the entire aorta. Extensive calcification involved the arch and descending portions of the aorta (Fig. 5). Direct: Intravenous aortograms showed severe arteriosclerotic changes of the entire aorta with elongation and widening. There was roentgenographic evidence of dissection of the posterior lateral wall of the descending thoracic aorta with evidence of contrast material within the wall in this area. The dissection could be followed to the level of the 12th thoracic vertebra. The impression was dissecting aneurysm of the descending thoracic aorta. No localized evidence of aneurysm in the upper abdominal aorta was seen (Fig. 6 and 7).

CASE III. This 77 year old white male entered the hospital with the chief complaint of abdominal pain and rectal bleeding. Clinical examination revealed a perirectal abscess. A history of angina pectoris was elicited.

Physical Examination. Physical examina-



FIG. 4. Case 1. Oblique intravenous angiocardiogram shows a channel within the wall of the aorta filled with contrast material. The lucency on either side of the channel is marked by arrows and represents inner and outer walls of the dissection.



Fig. 6. Case 11. Left anterior oblique intravenous aortogram shows some dilatation of the entire aorta with elongation and widening along the posterior wall of the descending thoracic aorta. Contrast material is noted within the wall. The dissection appears to begin distal to the left subclavian artery and can be followed down to the level of the diaphragm, with contrast material in the wall (arrows).

tion was unremarkable. Blood pressure was 110/80.

Roentgenologic Findings. Indirect: A roentgenogram of the abdomen revealed a linear calcification to the left of the lumbar spine, indicating a possible abdominal aortic aneurysm. A roentgenogram of the chest revealed some fullness of the aortic arch (Fig. 8). Direct: An intravenous aortogram demonstrated a severely tortuous and minimally dilated abdominal aorta with secondary arteriosclerotic disease. An intravenous aortogram visualizing the thoracic area showed the aortic arch to be widened. In the oblique projection, there was a definite appearance of contrast material within the wall of the aorta. The impression was chronic dissection of the aorta involving the ascending and proximal descending aorta. No operation was performed because the patient's cardiac status was unsatisfactory for a major thoracotomy (Fig. 9).

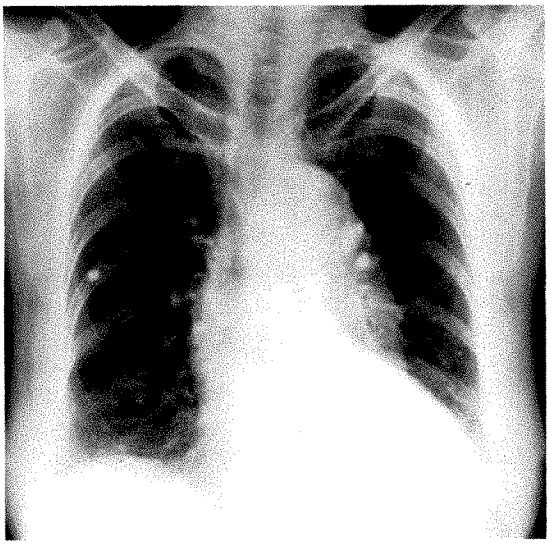


Fig. 5. Case 11. Frontal chest roentgenogram shows moderate dilatation of the entire aorta with extensive calcification involving the aortic arch.

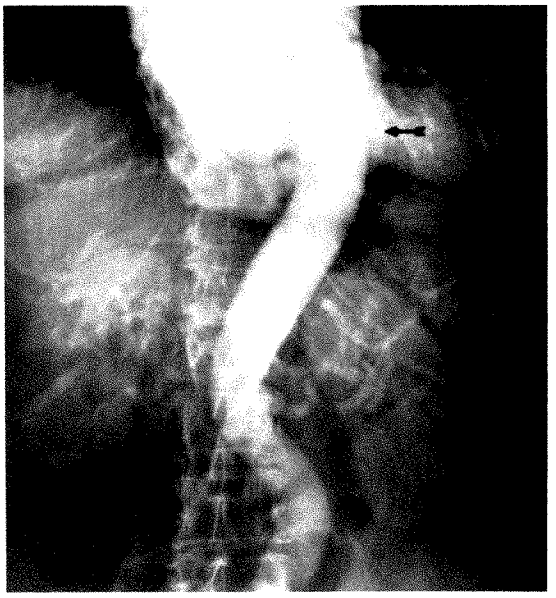


Fig. 7. Case 11. A second intravenous anteroposterior aortogram shows the lower portion of thoracic aorta, with the contrast material in the wall (arrow), well demonstrated to the level of the diaphragm.

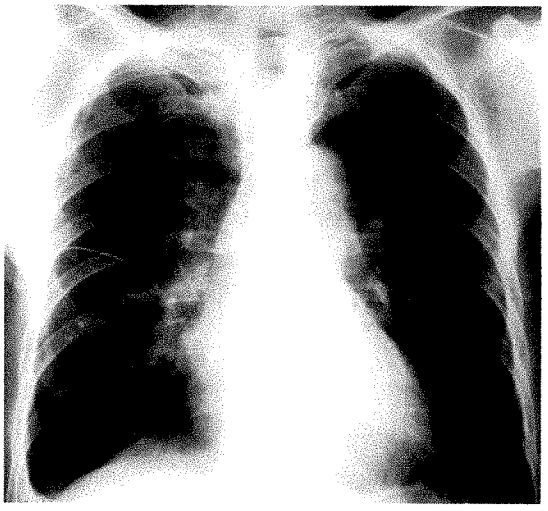


FIG. 8. Case III. Frontal chest roentgenogram shows an unfolded tortuous aorta with some widening of the aortic arch.

CASE IV. This patient was a 52 year old Negro married attorney who entered the hospital for the first time with a complaint of substernal pain, upper abdominal pain, and low back pain of approximately 2 hours' duration. The patient had noted generalized abdominal discomfort for the past 2 weeks, especially in the mornings. He had been well otherwise, until 2 hours prior to admission, when he had sudden onset of steady low back pain followed by generalized crampy lower abdominal pain and substernal pain accompanied by dyspnea.

Physical Examination. Physical examination revealed a Grade 1 systolic murmur at the apex. No other significant physical findings were noted.

Roentgenologic Findings. Indirect: Chest examination on admission revealed some linear strand-like densities involving both lower lung fields presumably on the basis of chronic fibrotic disease of the lungs. The cardiac silhouette was not striking. However, the aortic arch showed uniform dilatation and uncoiling, with prominence, particularly of the descending aorta (Fig. 10). Direct: An angiocardiographic examination of the aorta, both thoracic and abdominal, revealed a large dissection starting in the region of the aortic arch and ending abruptly at the diaphragmatic dome. A sharp angulation was noted as the thoracic aorta passed through the diaphragmatic dome. The roentgenographic impression was chronic dissection of the aorta involving only the thoracic aorta (Fig. 11 and 12).

Comment. The angiocardiographic investigation was initiated by the Department of Radiology. A tortuous dilated aorta was evident with associated mediastinal thickening at fluoroscopy (gastrointestinal study).

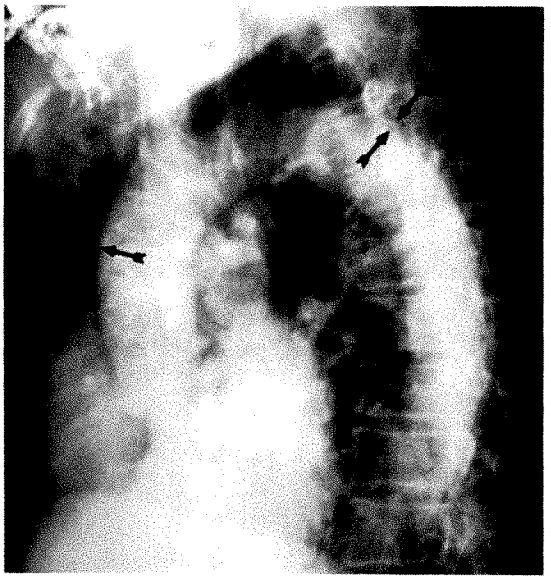


FIG. 9. Case III. Left anterior oblique intravenous aortogram shows a relatively normal aortic lumen with contrast material within the wall of the aorta (single arrow). The increased thickness of the aortic wall is indicated by the upper arrows.

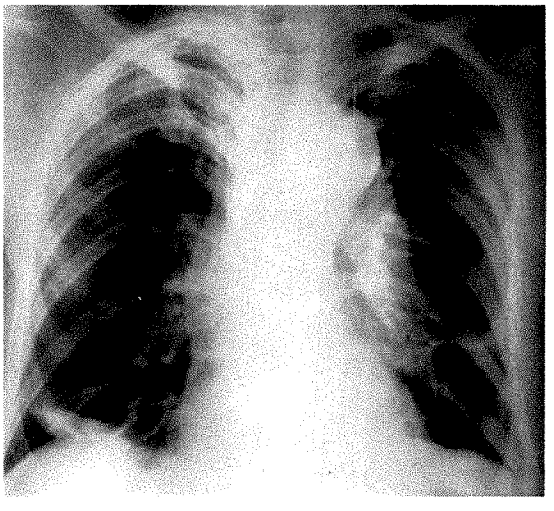


FIG. 10. Case IV. Frontal chest roentgenogram showing a relatively unremarkable cardiac silhouette. Uniform dilatation, uncoiling and prominence of the aorta is seen, particularly of the descending aorta.

Case v. This 37 year old Negro woman came to St. Luke's Hospital with a history of 2 years of exertional dyspnea and heart murmur. Five days prior to admission, the patient had developed chest pain and discomfort associated with paresthesias of the legs and right arm. Past history revealed that many of her family had died at early ages of cardiovascular disease and there were 2 cases of documented Marfan's syndrome.

Physical Examination. Physical examination revealed a blood pressure in the right arm of 80/50, in the left arm of 150/40 with a pulse of 130 and respirations of 24. There was a loud systolic diastolic murmur over the right anterior chest and sternal border. A_2 was absent. In the extremities, there were bounding pulses except in the right arm which was colder than the rest of the body.

Roentgenologic Findings. Indirect: A routine chest roentgenogram revealed a massively enlarged ascending aorta. There was left ventricular enlargement as well (Fig. 13). Direct: Intravenous angiocardiograms showed a pronounced enlargement of the heart with deformity of the cardiac chambers secondary to massive aneurysmal dilatation of the sinuses of Valsalva and the proximal aorta. The large aneurysm of the sinuses of Valsalva had a pronounced extrinsic pressure effect on the atrium and right ventricle which were compressed and displaced downward (Fig. 14). The right pulmonary artery was displaced upward but not markedly enlarged. The left atrium and left ventricle were markedly enlarged with prominence and dilatation of the left ventricle. The aneurysmal dilatation of the sinuses of Valsalva measured 12 cm. in width and extended for a distance of 14

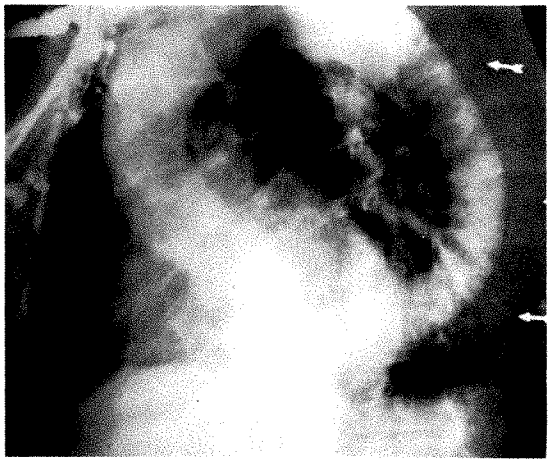


Fig. 11. Case iv. Left anterior oblique angiocardiogram shows a large dissection originating in the region of the aortic arch and extending down to the dome of the diaphragm. The marked thickness of the aortic wall is indicated by arrows.



Fig. 12. Case iv. Frontal angiocardiogram again shows the large soft tissue density outside the visualized aortic lumen. This represents aortic wall with the gross dissection widening the wall to almost the thickness of the normal lumen. The outer margin of the aortic shadow is marked by arrows. This projection shows the thickness of aortic wall returning to near normal at the level of the diaphragm.

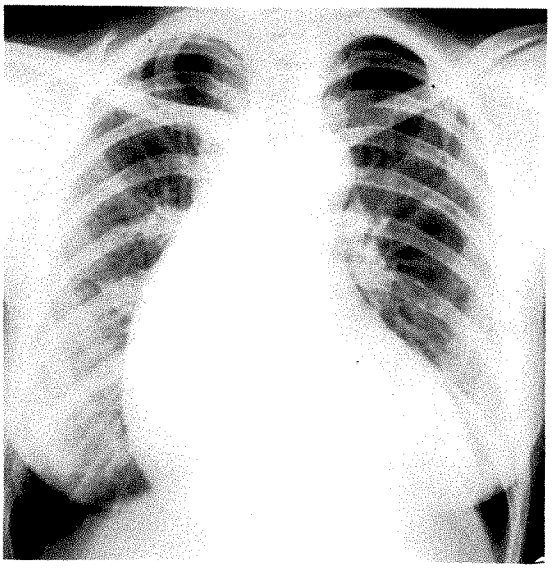


Fig. 13. Case v. Frontal chest roentgenogram shows a massively enlarged ascending aorta as well as left ventricular enlargement.

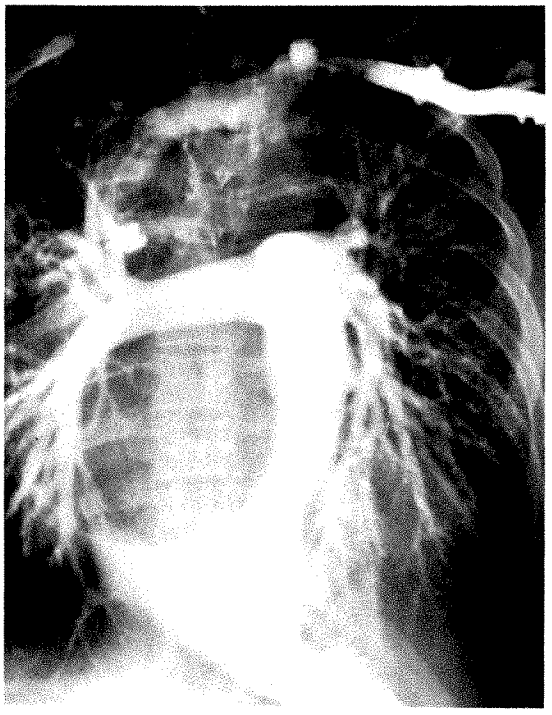


FIG. 14. Case v. Intravenous angiocardiogram shows marked extrinsic pressure upon the right atrium and right ventricle, which are compressed and displaced downward. Displacement of the outflow tract of the right ventricle is to the left. The right pulmonary artery is displaced upward minimally.

cm. (Fig. 15). The ascending aorta was displaced upward and there was buckling in the region of the transverse portion of the arch. After intravenous aortography, selective aortography by retrograde catheterization was performed (Fig. 16). Following injection at the level of the aneurysmal dilatation of the aorta, regurgitation was noted into the left ventricle. The aneurysm of the sinuses of Valsalva remained opacified for 9 seconds. Operation was not attempted. The patient died 11 days following admission.

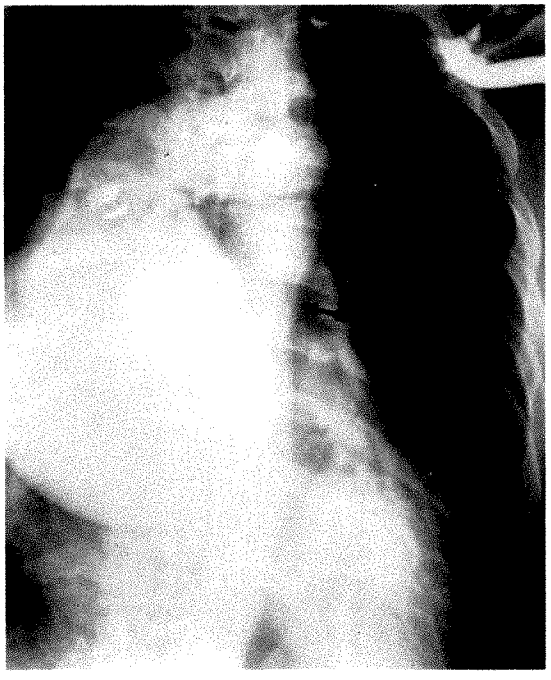


FIG. 15. Case v. Intravenous angiocardiogram shows massive aneurysmal dilatation of the sinuses of Valsalva and proximal aorta. The descending aorta reveals a lamination to its outer border (arrows) indicating contrast material within the wall of the dissection.

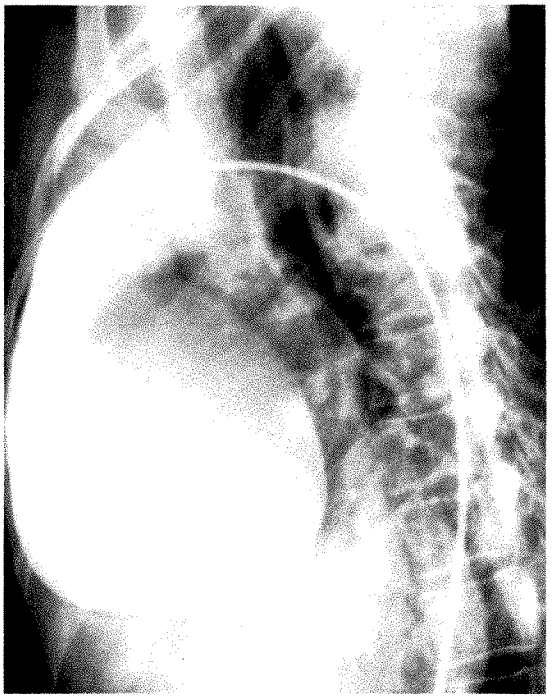


FIG. 16. Case v. Selective aortogram by means of retrograde catheterization shows contrast material pooling in the massively dilated aneurysm of the sinuses of Valsalva.

Pathology. At autopsy, a huge dissecting aortic aneurysm due to medial degeneration was demonstrated. There was degeneration of elastic tissue and destruction of vasa vasorum with hemorrhage and cystic change. In addition, there was dilatation and incompetence of the aortic valve ring and a large aneurysm of the ascending aorta. There were ruptures of both inner and outer walls of the aneurysm. The resulting cardiac tamponade was the cause of death. The aneurysm was opened and a dissection of the aorta with a smooth shiny surface completely encircling the lumen and extending from the aortic ring to the femoral arteries was seen. It was also noted to extend for a short distance into the great vessels of the arch of the aorta. The coronary arteries were spared but the dissection involved the bronchial and vertebral arteries (Fig. 17, 18 and 19).

Case vi. This 56 year old white man entered the hospital with the chief complaint of dizziness of 1 day's duration.



Fig. 17. Case v. Gross specimen of heart, aorta and great vessels. Arrow points to huge unopened dissecting aneurysmal sac.



Fig. 18. Case v. Opened dissecting aneurysm (cross section). False channel is marked with arrow.

Physical Examination. Blood pressure was 260/150 in the right arm and 235/135 in the left arm. There was a Grade 3, harsh, systolic murmur. Clinical impression was hypertension and possible dissection of aorta.

Roentgenologic Findings. Indirect: Examination of the chest revealed the cardiac size to be at the upper limits of normal. There was widening of the ascending and descending limbs of the aorta. A double density at the aortic arch suggested dissection of the aorta (Fig. 20). One week later, a cardiac fluoroscopy showed the ascending aorta's pulsations to have diminished and a cardiac esophagram strongly suggested a double contour. Direct: Intravenous aortogram revealed a definite increase in thickness of the wall of the aorta, indicating dissection within the wall. There was a very striking double density demonstrated at the base of the aorta continuing through the region of the apex of the aorta and for a distance of approximately 7 cm. inferiorly along the left aortic border. This was interpreted as contrast material within a large dissection. The impression was dissection about

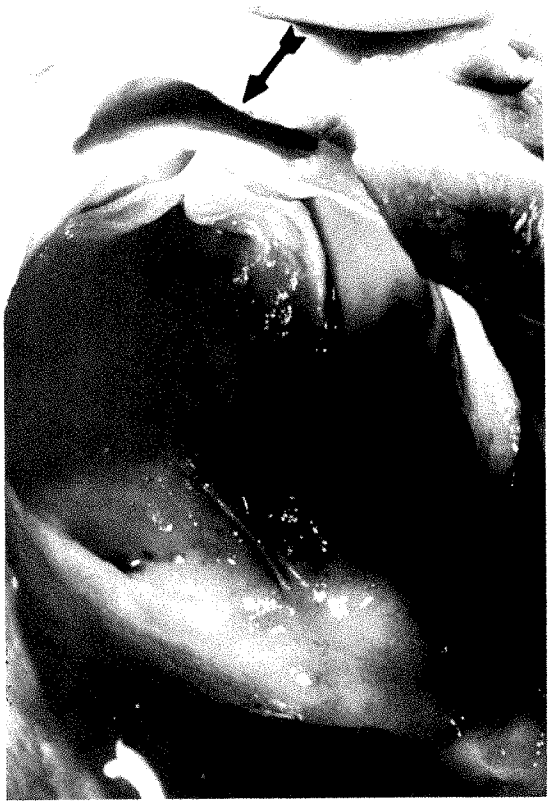


FIG. 19. Case v. False channel visualized from above (arrow), looking into dilated aortic valve.

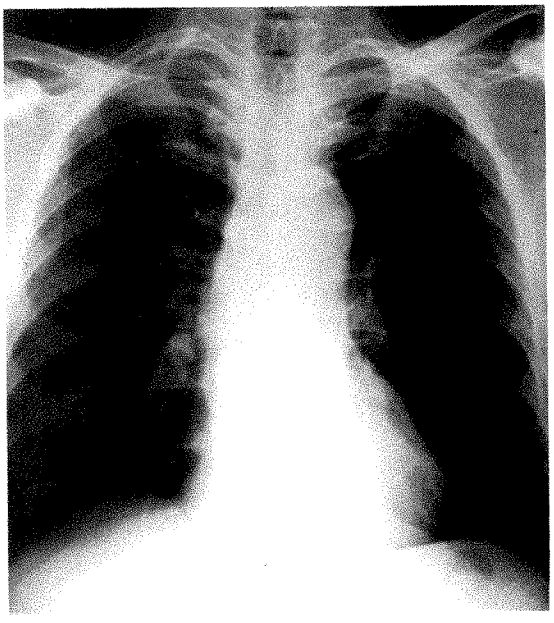


FIG. 20. Case VI. Chest roentgenogram showing widening of the ascending and descending limbs of the aorta.

the aortic arch involving the descending aorta as well (Fig. 21).

CASE VII. This was the first admission of a 62 year old white woman who had well documented hypertension for many years. Her blood pressure had been approximately 220/110. On the morning of admission she developed acute chest pain radiating to the front of her chest. The pain gradually migrated to the upper epigastric area. On presentation to the hospital, she appeared in acute distress.

Physical Examination. Shortly before admission her blood pressure was measured at 130/60. Physical examination was otherwise essentially unremarkable.

Roentgenologic Findings. Indirect: Chest roentgenogram revealed a markedly prominent, elongated aorta with calcification of the aortic knob. The soft tissue outlining the aorta extended beyond the calcification. The possibility of dissection was mentioned. Comparison with previous chest roentgenograms showed that the aortic arch had widened (Fig. 22 and 23). Direct: An intravenous aortogram showed widening of the descending aorta just distal to

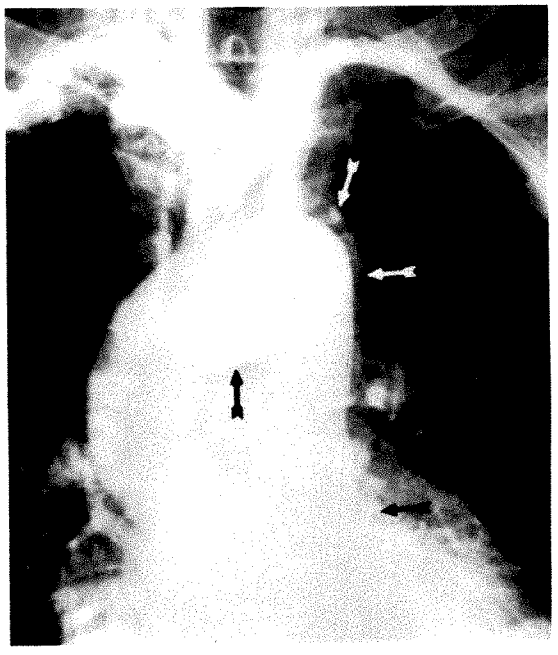


FIG. 21. Case VI. Intravenous aortogram demonstrating a double density, starting at the base of the aorta and rising through the apex of the aorta and then descending along the thoracic aorta (upper black arrow). Note thickness of the wall of the aorta (white arrows and lower black arrow).

the origin of the left subclavian artery. Aneurysmal dilatation occurred at this region. Dissection of the aorta as manifested by a thickened aortic wall began at this level, just distal to the left subclavian artery, and was followed as far as the level of the diaphragm. The exact point of re-entry could not be identified. However, the thickness of the aortic wall decreased as the aorta passed through the diaphragm (Fig. 24).

Case VIII. This was the first St. Luke's Hospital admission of a 60 year old man who was referred with a diagnosis of dissection of the thoracic aorta, following aortography performed at another hospital. The patient presented with symptoms of severe upper abdominal pain described as "tearing" in nature.

Physical Examination. Prior to admission to the hospital, the blood pressure was 200/78. His peripheral pulses were normal. On admission to the hospital, his blood pressure had dropped to 155/95. He was in no acute distress. However, he experienced lower thoracic and left lower quadrant abdominal soreness. No murmurs were heard.

Roentgenologic Findings. Indirect: Chest examination revealed widening of the mediastinum with a prominent aortic knob and a mass in the left hilar area which was continuous with the aortic shadow (Fig. 25). Direct: Angio-

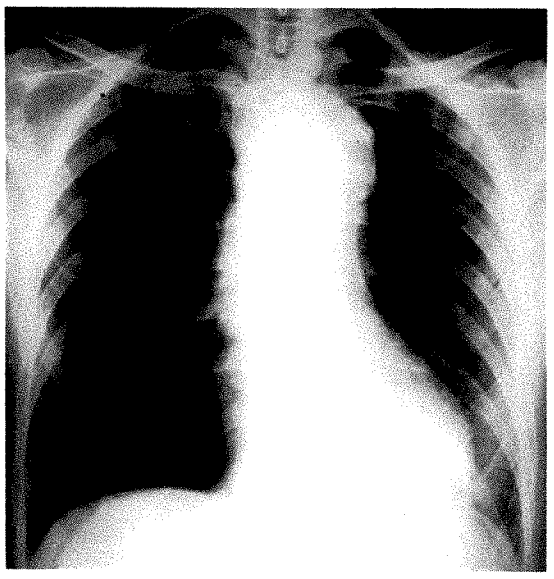


Fig. 22. Case VII. Chest roentgenogram prior to admission showing an arteriosclerotic unfolded aorta with calcification.

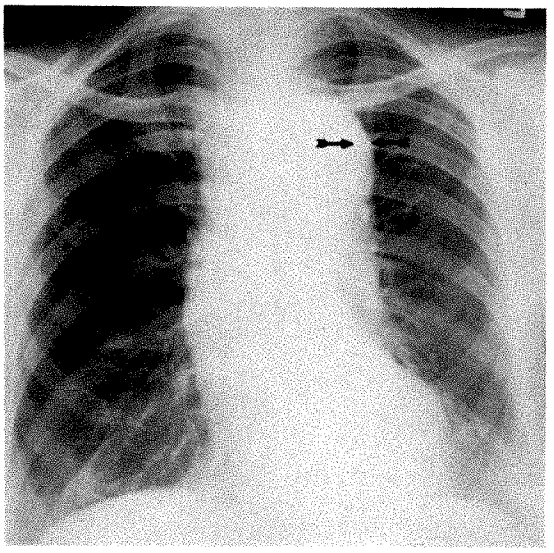


Fig. 23. Case VII. Interval chest roentgenogram showing an increase in the dimensions of the aortic arch with an increase in soft tissue adjacent to the calcification (arrows).

cardiograms revealed aneurysmal dilatation of the descending portion of the aorta with definite thickening of the wall of the aorta, prin-

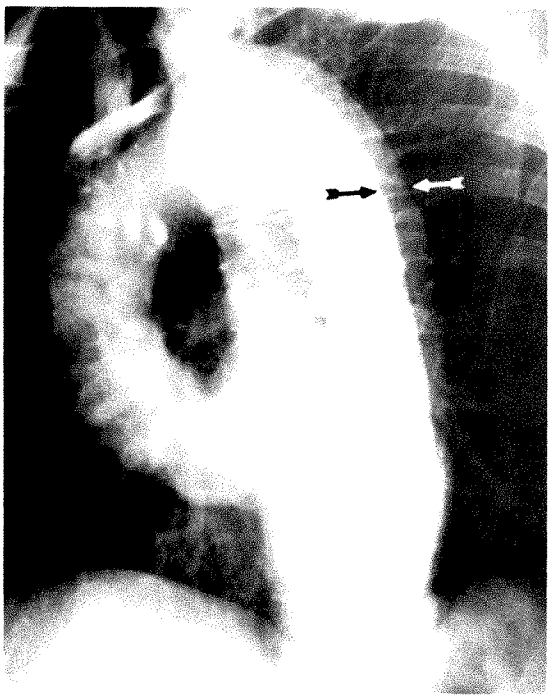


Fig. 24. Case VII. Intravenous aortogram demonstrating marked increased thickness of the aortic wall and the aneurysmal dilatation of the arch of the aorta. Diagnosis—dissecting aneurysm.

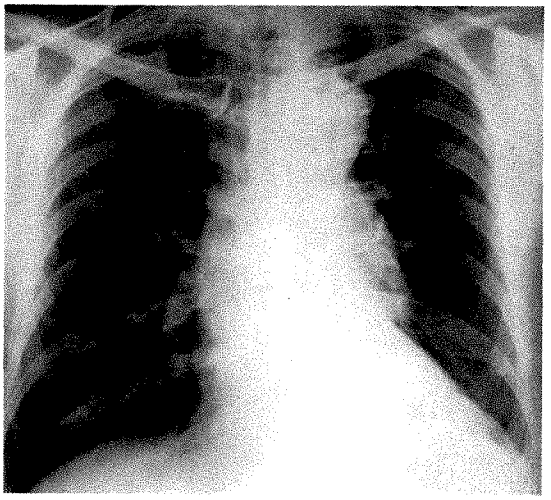


FIG. 25. Case VIII. Frontal chest roentgenogram showing pronounced widening of the aortic arch and the descending aorta.

cipally in the distal aortic arch area (Fig. 26).

Operative Findings. A transthoracic aortic aneurysmorrhaphy was performed. The aortic arch and descending aorta were dilated and a soft area in the mid-thoracic region appeared weak and was repaired with 3 suture lines. The patient died shortly after operation.

Pathology. At autopsy, the aorta showed an intact intima. The media was thickened and contained large vascular channels with numerous red blood cells and some extravasation. There was an acute and chronic inflammatory response about the vascular channels of the outer media. A portion of the wall of the media demonstrated evidence of degeneration with fragmentation and hyalinization. The media contained a secondary channel. A laminated thrombus filled a large portion of the vascular channels within the media.

Diagnosis. Dissection of the thoracic aorta with aneurysm formation. The patient died as a result of a left pleural hemorrhage, secondary to operation and bilateral bronchopneumonia.

CASE IX. This was the first St. Luke's Hospital admission of a 59 year old Negro man who entered with the chief complaint of back pain. Two days prior to this he developed sudden pain in the left leg followed by low back and mid-scapula pain associated with left anterior chest pain and generalized weakness. The pain subsided after 36 hours but low back pain persisted.

Physical Examination. On admission, blood pressure in the right arm was 245/140, left arm 245/140, pulse 84, respirations 16. There was a Grade 1 systolic murmur audible at the base, to the right of the sternum, and not transmitted to the neck. Mazzini was 4 plus, VDRL was positive undiluted.

Roentgenologic Findings. Indirect: Frontal chest roentgenogram revealed a prominent ascending aorta (Fig. 27). Direct: Intravenous aortogram showed no definite dilatation of the aortic arch. However, at the takeoff of the innominate artery, contrast material could be delineated dissecting its way out into an abnormal channel which ran along the lateral portion of the thoracic aorta. It measured approximately 0.6 cm. The abnormal channel extended down to the dome of the diaphragm. Roentgenographically, the diagnosis of dissection of the aorta was established (Fig. 28).

CASE X. This 48 year old woman presented at the hospital with deep pain under her left breast which occurred suddenly. On occasion it radiated to the left lumbar area.

Physical Examination. Blood pressure was 150/80. Patient had long fingers and a high arched palate and a family history of one parent dying of a perforated aorta.

Roentgenologic Findings. Indirect: Frontal

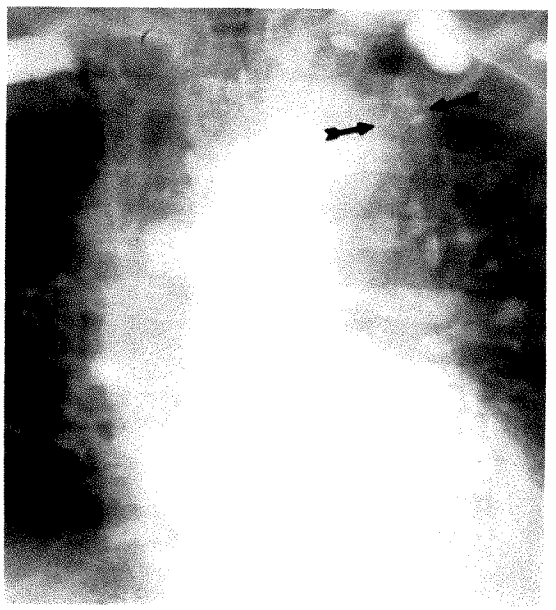


FIG. 26. Case VIII. Angiocardiogram shows aneurysmal dilatation of the arch and descending aorta with marked thickening of the wall of the aorta, indicating a dissecting aneurysm. (Courtesy of David Seibert, M.D., Pt. Pleasant, N. J.)

chest roentgenogram showed an aorta which was normal in size (Fig. 29). Direct: Intravenous aortograms were interpreted as showing a dissection beginning in the transverse portion of the arch of the aorta. The exact point of reentry could not be evaluated (Fig. 30 and 31).

Operative Findings. At operation, the aorta was opened and explored thoroughly. No dissection was seen.

Comment. Although the aortic channel was normal in appearance, contrast material was thought to be outside the lumen of the aorta. This area measured 3 mm. Because this case was clinically thought to be a forme fruste of Marfan's syndrome, operative intervention was undertaken. There is no explanation for the normal appearing aorta at operation.

It is possible that adventitial vascular opacification may imitate a dissecting aneurysm.

Similar lack of confirmation of supposed aortic dissection diagnosed by contrast studies has been encountered at autopsy and at surgery.

Case xi. This 72 year old white man was well until 3 years prior to admission when he was seen in mild congestive failure with moderate hypertension. Eight hours prior to this present admission, he developed acute middorsal and lumbodorsal pain. The pain was associated with weakness but apparently not unconsciousness. Shortly after the onset of pain, there was substernal radiation into both shoulders and the left arm. The patient denied previous chest pain.

Physical Examination. On admission to the hospital the patient's blood pressure was

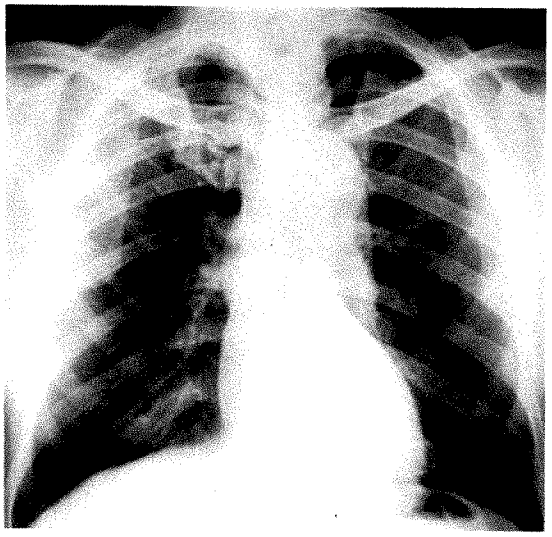


Fig. 27. Case ix. Frontal chest roentgenogram showing some fullness of the aortic arch.

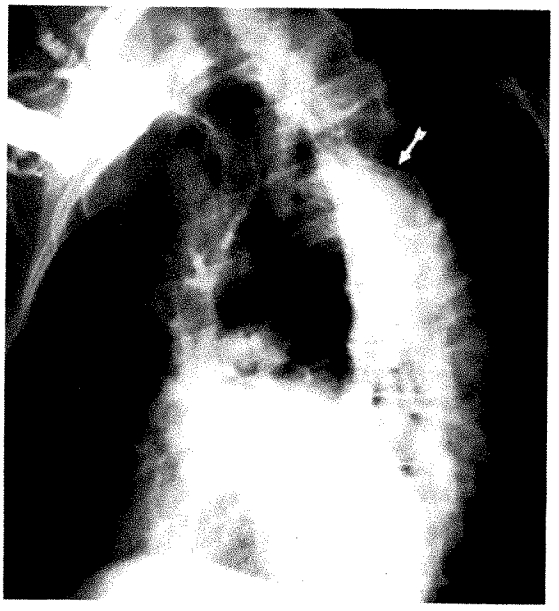


Fig. 28. Case ix. Intravenous aortogram shows no striking dilatation of the aortic arch; however, contrast material is seen dissecting into an abnormal channel which runs along the lateral portion of the descending aorta. The channel measures approximately 6 mm. in width (white arrow).

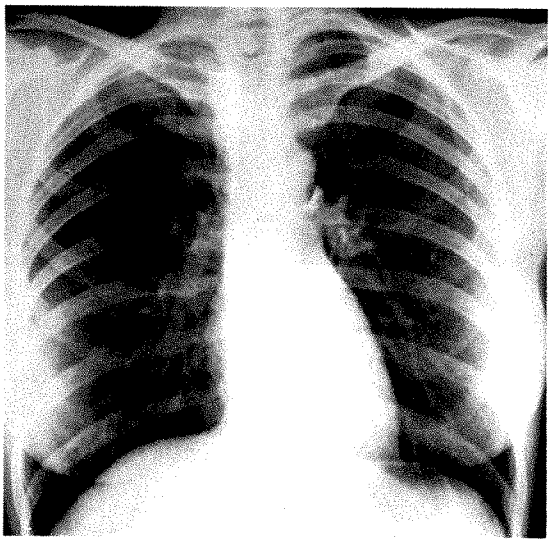


Fig. 29. Case x. Frontal chest roentgenogram reveals a normal aortic arch. The cardiac silhouette is normal.

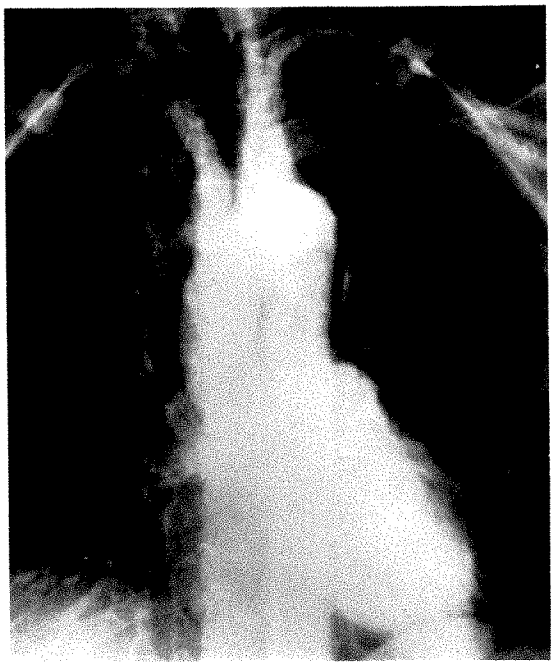


FIG. 30. Case X. Frontal intravenous aortogram shows a normal aortic arch with normal great vessels and a normal descending aorta.

260/120, with a pulse of 66. There was a loud, booming, Grade 4, harsh, systolic murmur with poor radiation. Clinical impression was dissection of the aorta with aneurysm, and mild congestive failure.

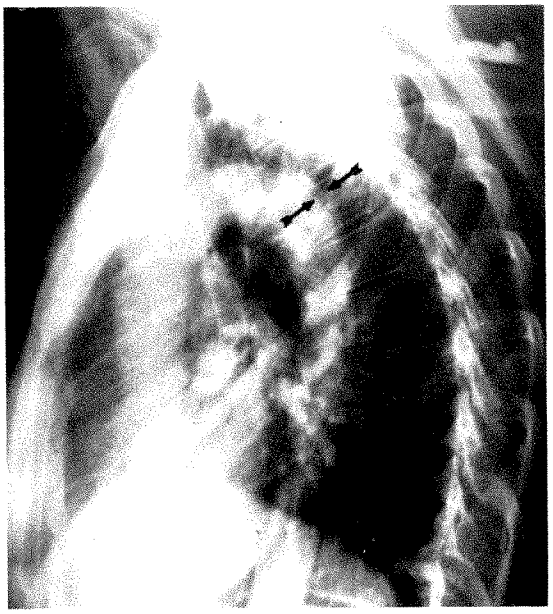


FIG. 31. Case X. Left anterior oblique intravenous aortogram shows contrast material in the aortic wall outside the main lumen (arrows). This was interpreted as a dissection of thoracic aorta.

Roentgenologic Findings. Indirect: Roentgenograms revealed a moderate degree of cardiomegaly with prominence of the left ventricle. The aortic arch and proximal portion of the descending aorta were markedly enlarged. Comparison with a roentgenogram taken 2 years prior to admission showed a significant increase in the width of the descending aorta and arch (Fig. 32 and 33).

Comment. Angiocardiographic investigation was not pursued because of the patient's age and clinical improvement. Sixteen months have elapsed since the initial episode and the patient is alive and well.

CASE XII. This was the fourth hospital admission of a 71 year old white man with the chief complaint of chest pain of 3 hours' duration. Early on the morning of admission, the patient awoke with severe anterior chest pain associated with weakness and sweating.

Physical Examination. Physical examination revealed a blood pressure of 140/80, with a pulse of 60, and respiration of 12. The patient was perspiring profusely and complaining of left anterior chest pain. Forty-eight hours following admission the patient developed respiratory distress and died.

Roentgenologic Findings. Indirect: Roentgenogram 1 year before admission showed a relatively normal chest (Fig. 34). A portable

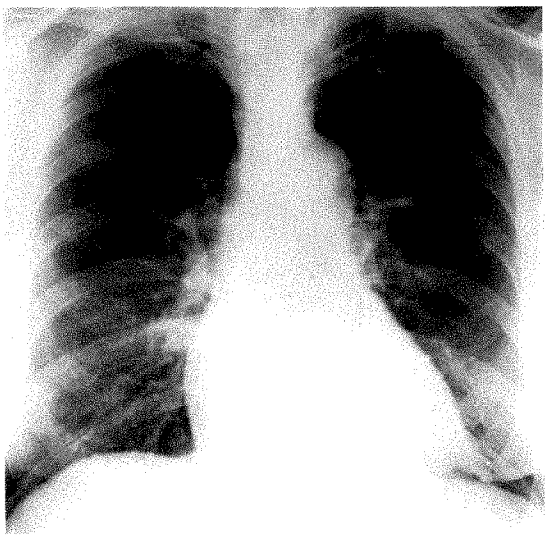


FIG. 32. Case XI. Frontal chest roentgenogram shows cardiomegaly and fullness of the aortic arch secondary to arteriosclerotic unfolding of the aorta.

chest roentgenogram prior to death showed widening of the aortic arch and dilatation of the brachiocephalic vessels (Fig. 35).

Pathology. At postmortem examination, there was incomplete separation of the media in its outer third with a large collection of red blood cells in these spaces. In one area, the media had been split at multiple levels. In several sections there were mucoid areas replacing muscle fibers.

Diagnosis. Medionecrosis of the aorta with dissecting aneurysm of the thoracic aorta and abdominal aorta.

Case xiii. This 76 year old white man was admitted to the hospital with a history of pain in the left neck and shoulder, which started on the morning of admission and increased in intensity within the next 2 hours.

Physical Examination. On admission to the hospital, physical examination revealed a cold patient in shock with a respiratory rate of 20, and unobtainable pulse and blood pressure. Emergency shock therapy was instituted. However, shortly after admission the patient died.

Roentgenologic Findings. Indirect: Examination of the chest prior to admission revealed some arteriosclerotic change of the aorta and no enlargement of the cardiac silhouette. A roentgenogram taken immediately prior to death showed no interval change in the chest.

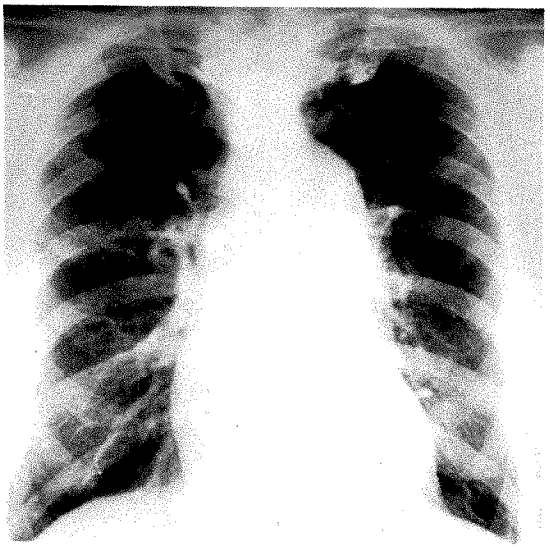


Fig. 33. Case xi. Chest roentgenogram immediately prior to admission (3 years after Fig. 32) to the hospital shows marked widening of the aortic arch with some obscuring of the pulmonary artery by the descending aorta.

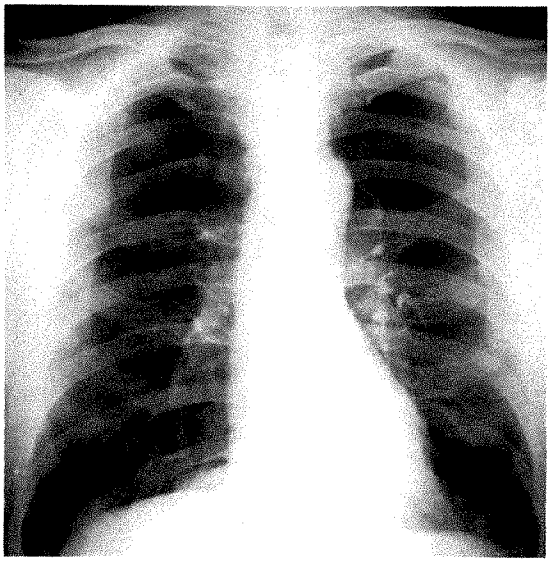


Fig. 34. Case xii. Posteroanterior chest roentgenogram 1 year prior to death showing no gross dilatation of the aortic arch.

No widening of the aortic arch had occurred (Fig. 36).

Pathology. The aorta showed marked calcification and fibrosis of the intima. There were fresh red blood cells and recent thrombi dissecting the outer third of the media. The elastic fibers of the media were swollen and fragmented. Primary lesions on pathology were considered to be general arteriosclerosis and medial

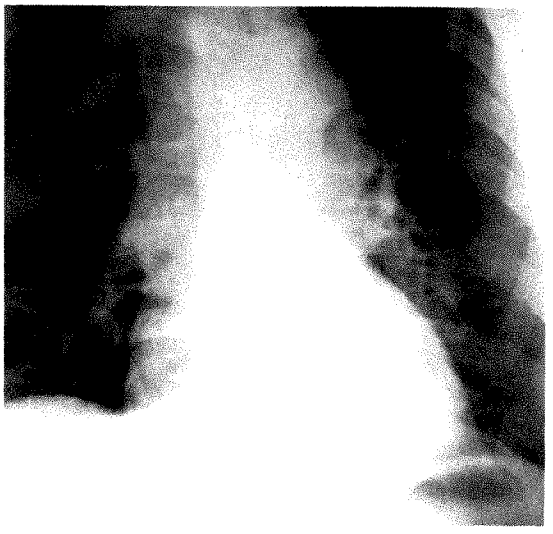


Fig. 35. Case xii. Chest roentgenogram prior to death showing considerable dilatation of the ascending arch of the aorta and descending aorta.

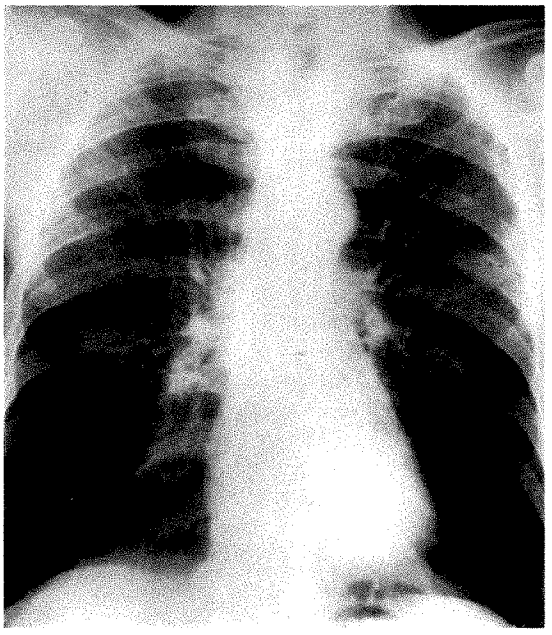


FIG. 36. Case XIII. Chest roentgenogram immediately prior to death reveals arteriosclerotic change of the aorta with tortuosity but no striking enlargement. This study when compared to previous roentgenograms showed no change in the dimensions of the aortic arch.

necrosis of the aorta. Secondary terminal lesions of dissecting aneurysm and hemopericardium were described.

Comment. Although unusual, the aorta may appear normal roentgenographically and yet a dissection may be present.

CASE XIV. This 72 year old white man was admitted to the hospital on December 23, 1962 because of severe, sharp chest pain which radiated to the central portion of the back. While being examined by his physician 2 weeks prior to this admission, he became paralyzed from the nipples down. Paralysis receded over the next few days during which time there was recurrent chest pain, less severe in degree.

Physical Examination. On admission the patient's blood pressure in the right arm was 170/90 and in the left arm 150/70. The temperature was 102°F., respirations 20, pulse 108. The patient was alert and in no distress. He appeared to be in exceptionally good health for his stated age. No murmurs were evident on auscultation, and good pulses were present in all four extremities.

Roentgenologic Findings. Indirect: Examination of the chest in the posteroanterior and left anterior oblique positions revealed a considerable increase in the over-all dimensions of the descending thoracic aorta. A distance of approximately 1 cm. was present between the calcification in the aortic knob and the most lateral aspect of the aorta in this region. The conventional roentgenograms were very suggestive of a dissecting aneurysm (Fig. 37 and 38). Direct: The following morning intravenous aortograms were made both in the anteroposterior and left anterior oblique projections. An extremely large dissecting aneurysm of the thoracic aorta was well demonstrated which appeared to originate at or distal to the left subclavian artery. The point of reentry was immediately above the diaphragmatic dome. The area of maximum dissection was just distal to the aortic knob and measured 4 to 5 cm. The true lumen of the descending thoracic aorta was impinged upon anteriorly and decreased in transverse dimensions. Poor definition of the outer margins of the dissection below the aortic knob was highly suggestive of a continuing active process. Figures 39 and 40 demonstrate the dimensions of the dissection.

Comment. In view of the presence of continuing chest pain and the unequal blood pressures in both arms together with the lack of angiocardiographic demonstration of the outer margins of the dissection immediately below the aortic knob, the clinical and roentgenographic impression was that an active process was present. For this reason, the patient was

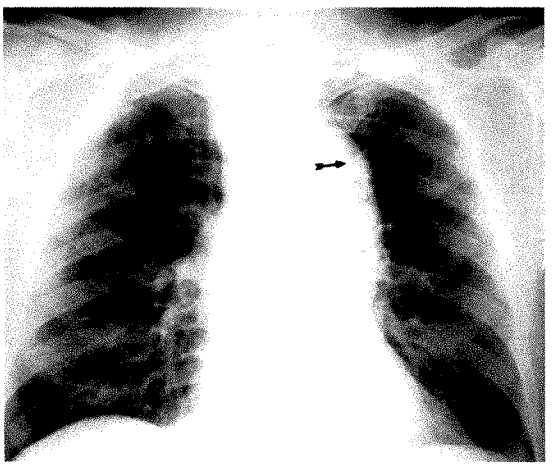


FIG. 37. Case XIV. Posteroanterior chest roentgenogram shows considerable widening of the descending thoracic aorta. A distance of 1 cm. was present between the calcification in the aortic knob and the left lateral aspects of the aorta (arrow).

operated on the following day and a large dissection originating at the left subclavian artery and extending to the diaphragmatic dome was seen. Approximately 6 inches of the aorta was removed immediately distal to the left subclavian and replaced by a dacron graft. Figure 41 is the surgical specimen in cross section, photographed at the time of surgery. The patient is alive and well and was discharged from the hospital on January 26, 1963.

Case xv. This 75 year old Negro man was admitted to the hospital on December 30, 1962 because of urinary retention. Other than occasional insignificant chest pains in past years, he had always been in good health.

Physical Examination. Physical examination revealed a patient in no distress who had lower abdominal discomfort. Pulse and respirations were normal. Blood pressure in the right arm was 170/90 and in the left arm 200/115. A Grade 2 systolic murmur was present at the apex. No former studies were available for comparison.

Roentgenologic Findings. Indirect: A conventional posteroanterior study of the chest revealed a considerable increase in the transverse cardiac dimensions with prominence of the left ventricle. A large soft tissue mass was present in the right paratracheal region and measured 6 cm. in diameter. The lung fields were clear (Fig. 42). Fluoroscopy revealed this mass to be continuous with the arch of the aorta and to pulsate. The appearance was suggestive of an aneurysm of the innominate artery. Direct: Intravenous angiocardiograms in the antero-

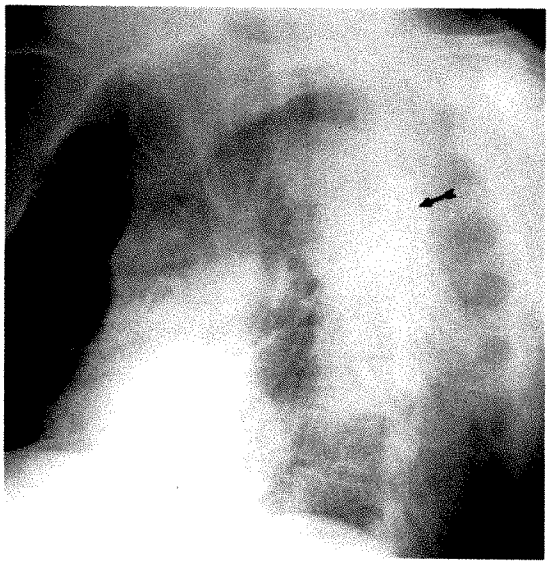


Fig. 38. Case xiv. Left anterior oblique study showing lateral margin of the descending thoracic aorta (arrow).

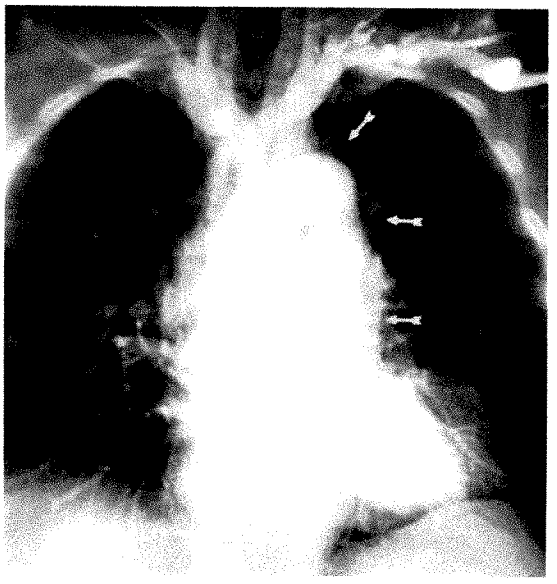


Fig. 39. Case xiv. Anteroposterior intravenous aortogram showing lateral wall of false channel (arrows). It measured 4–5 cm. in diameter and was seen to best advantage on the left anterior oblique study.

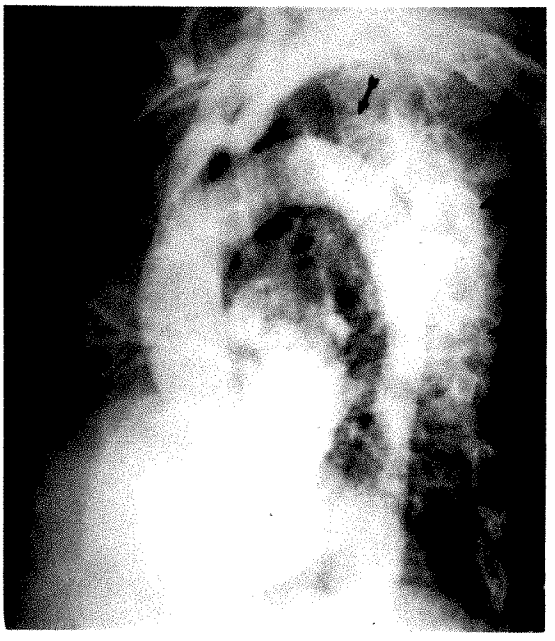


Fig. 40. Case xiv. Left anterior oblique intravenous aortogram showing the false channel. The lateral wall of the dissection is poorly defined (arrows). The true lumen of the aorta is impinged upon anteriorly.

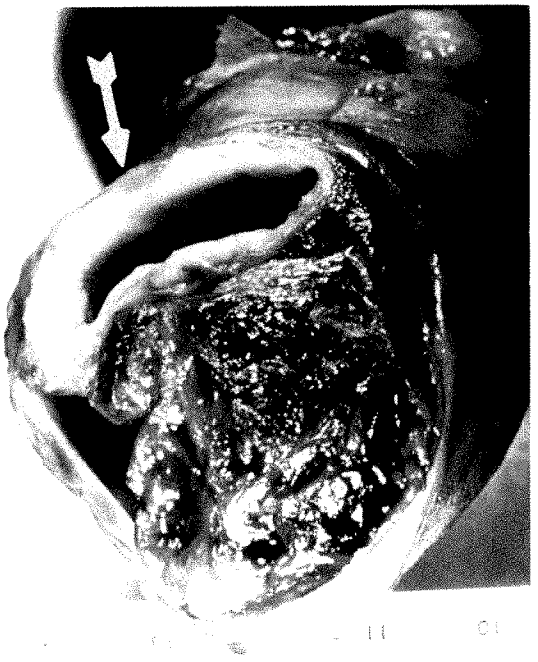


FIG. 41. Case XIV. Surgical specimen of aorta distal to left subclavian artery, seen in cross section. Arrow indicates true lumen of aorta which is narrowed by huge dissection adjacent to it.

posterior projection revealed the mass in the right paratracheal region to be due to a dissecting aneurysm of the innominate artery. Only the central portion, *i.e.*, 2 cm. of this vessel opacified during the procedure. The thoracic aorta was well opacified and was extremely tortuous. The distance between the opacified lumen of the thoracic aorta and its outer margin was 8 to 9 mm. The findings were indicative of a dissecting aneurysm of the aorta involving not only the thoracic aorta but the innominate artery as well (Fig. 43, *A* and *B*). Approximately 4 cm. above the diaphragmatic dome the false lumen decreased in dimensions and at the diaphragmatic dome proper the dissection was no longer apparent, indicating re-entry in this region (Fig. 43*B*).

Comment. In view of the patients lack of complaints referable to the cardiovascular system and his response to urologic therapy, he was discharged 3 weeks after admission and is being followed in the cardiovascular clinic.

DISCUSSION

Although the history of dissecting aneurysm of the aorta dates back to the sixteenth century, less than 1,500 cases were reported in the literature up to 1960.[4] The youngest was an infant 3 months of age and the oldest was a woman 100 years old.[10] The autopsied cases varied from 0.1 per cent to 1 per cent.[3,12,17,21] Our present series of 15 cases, all of which had roentgen studies, was seen during the last 24 months. Angiocardiographic visualization of the dissection was obtained in 12 of these cases.

The underlying pathology can be essentially divided into two patterns. The first and most common is the widespread necrosis of the media of the aorta, often associated with extensive dissection and multiple channels which may be demonstrable angiocardiographically. The other is the arteriosclerotic type associated with marked changes secondary to arteriosclerosis involving both the intima and media. The media, which consists of elastic fibrils and compact layers of smooth muscle, is primarily responsible for the strength and elasticity of the aorta.[12]

A small tear through the intima and adjacent media permits the passage of aortic blood which is pumped at high pressure into the vessel wall.[11] This occurs

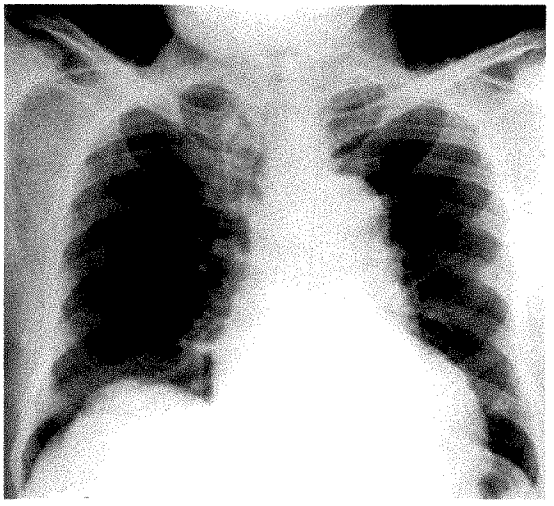


FIG. 42. Case XV. Posteroanterior chest roentgenogram showing the enlarged heart with a large soft tissue mass in the right paratracheal region, measuring 6 cm. in diameter.

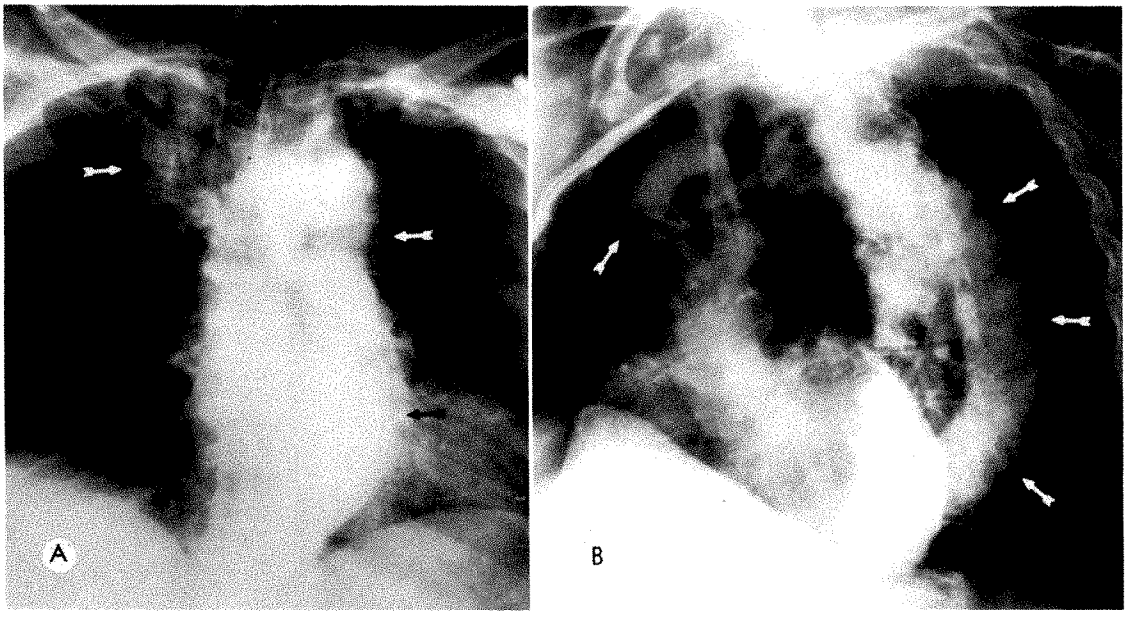


Fig. 43. Case xv. (*A*) Intravenous anteroposterior and (*B*) left anterior oblique angiocardiograms showing dissecting aneurysm of the innominate artery and aorta. The central portion of the paratracheal mass, true lumen of the innominate artery, measured 2 cm., the false lumen 4 cm. The wall of the descending thoracic aorta was 8–9 mm. in diameter. Re-entry was evident at the diaphragmatic dome (lowest arrow in *B*).

most often a few centimeters above the aortic valves or, less often, in the descending aorta just distal to the origin of the left subclavian artery.[8] Re-entry, when it does occur, is at the sites of physiologic obstruction, *i.e.*, the diaphragm or bifurcation of the abdominal aorta.

The correlation of the pathologic process with the varied and sometimes bizarre clinical picture can be better understood if we remember that the dissecting process may involve and extend along any or all of the aortic branches. The regional vascular supply may, in fact, not only be diminished but completely occluded.

The following clinical conditions have been related to dissecting thoracic aneurysms: Marfan's syndrome, hypertension, arteriosclerosis, pregnancy, aortic stenosis, coarctation of the aorta and trauma. Experimentally, it has been found that a toxic factor in sweet peas when fed to rats will produce scoliosis as well as medionecrosis of the arch of the aorta and subsequent dissection.[2]

Only in the last two decades have dissecting aneurysms of the aorta been recognized as the most frequent cardiac manifestation of Marfan's syndrome.[1] McKusic[16] discussed in detail the pathology of the aorta and major vessels as well as the allied abnormalities of the cardiovascular system associated with this disorder.

The relationship of pregnancy to thoracic dissections of the aorta is of interest.[15,20] Out of 49 women below 40 years of age, in 24, aortic dissections occurred during pregnancy.[15] Most of these cases were found in primiparas during the third trimester, unrelated to labor.

Hypertension is by far the most common associated finding. It occurs in from 50 to 80 per cent of the patients.

The clinical features are varied and are related directly to the extent of the dissection and the degree of involvement of specific visceral and parietal arteries. Fifty to 70 per cent of the patients are men, most of whom are over the age of 50 years. By far, the largest group of younger individuals

are those in whom medial necrosis is related to Marfan's syndrome.

Death occurred in 80–90 per cent of reported patients due to rupture of the aorta into the pericardial, thoracic or abdominal cavity.[18,21] Ten to 20 per cent survive the initial dissection but one-half of these patients die at a later date as a result of external rupture.[17] The patients who survive are of the chronic type with a spontaneously formed site of re-entry.[7] This is the so called "double barreled" aorta[5] (Case II).

Characteristically, there is a severe, tearing pain in the chest which may follow the path of the dissection. Syncope occurs in 10 per cent of the cases. Dyspnea, pallor and unconsciousness may follow. Unequal pulses may be present in the upper or lower extremities. Other physical findings are related to the specific vessel involved. Paraplegia is related to involvement of the anterior spinal artery. Proptosis and Horner's syndrome, secondary to occlusion of the great vessels, may also occur. Moersch and Sayre[17] reported a series of 12 cases of which almost half had neurologic signs due to interference with the blood supply of the spinal cord or brain. Pulsation of the sternoclavicular joint[13] or in the inguinal region may be an isolated finding.

Physical findings may include an aortic diastolic murmur which has been noted in roughly 25 per cent of the cases. A pleural effusion, if present, is almost always on the left side. Displacement of the trachea or esophagus can be cause by contiguous pressure from the enlarged aorta.

The most characteristic roentgenographic finding is a definite change in the diameter of the aorta during a short interval between 2 successive examinations.[22] The contour of the aorta is often irregular in the area of dissection,[11] with a resultant loss of sharpness of the aortic arch.[10] This may be associated with an increase in the diameter of the great vessels at their origins. Calcification in the aortic knob often assists in assessing the thickness of the aortic wall. The normal width of the aortic wall measures 2–3 mm.[10,12]

One other finding is obliteration of the notch below the aortic knob. A change in the diameter of the aorta (decrease) at the level of the diaphragm (Case IV) may be indicative of re-entry. However, a definite diagnosis prior to surgery can only be made by means of contrast visualization.[7,18] The first case diagnosed by angiocardiography[19] was later documented at surgery.[6]

The discovery of a larger number of cases followed by conclusive angiocardiographic demonstration is due to the development of simpler and more practical methods of intravenous contrast roentgenography. For visualization of either the thoracic or abdominal aorta, we have employed a method which is simple and reveals good detail of the aorta and false channels.[9] This technical development, plus the fact that intravenous angiocardiography is possible in spite of occlusive pathology or the patient's age, permits a rapid and definitive study prior to surgical intervention.

Visualization of the aorta by means of intravenous angiocardiography may reveal one, or several findings, if a dissecting aneurysm exists. There may be irregular narrowing of the main aortic channel. This was seen in only 1 of our cases. This may be associated with widening of the wall of the aorta. Less commonly the false channel is demonstrated.[7] This is usually separated from the true lumen by a thin translucent line[10] (Case I, II and IX). It is extremely important to determine the exact confines of the dissection and to localize the site of re-entry, if present.

In general, we are opposed to the routine use of the retrograde catheter technique in this disease. In at least 10 per cent of patients in the older age group, the retrograde passage of a catheter into the arch of the aorta is not anatomically possible. In addition, the dissection may extend along the aortic bifurcation[14] and the catheter may either be passed into the false lumen

or may not pass at all. There is no question that anatomic detail is better but this must be evaluated against the potential of increasing the dimensions of the dissection. We employed this route in Case v because of the unusual roentgen findings and the marked delay in emptying of the proximal aorta.

Translumbar aortography may in itself be responsible for the initiation of a dissection of the aorta. We feel that it has no place in the contrast studies of dissecting aneurysms of the aorta.

Early papers on surgical intervention and correction of the anatomic abnormalities associated with dissecting aneurysms are worthy of study.[3,5] The favored surgical procedures are local resection or the creation of an artificial site of re-entry. If a small area of the aorta is involved, it may be possible to replace the diseased portion with a graft. If a larger area is involved, the surgeon may reconstruct the aorta so that both lumens, the true and false, empty into one distal channel.

In view of the still high operative mortality and the long duration of survival following a natural re-entry, it is well to evaluate the role of surgery in cases where a site of re-entry can be demonstrated by means of angiocardiography.

SUMMARY

Dissection of the thoracic aorta, particularly of the chronic type, implying natural re-entry, is far more common than hitherto appreciated. Close cooperation between the clinician and the radiologist, together with awareness of the related pathologic processes, will permit earlier diagnosis.

Although conventional roentgen studies may raise a high level of suspicion, the ultimate diagnosis of thoracic dissecting aneurysms prior to surgery rests with the contrast demonstration of the false channels, increase in the thickness of the aortic wall and decrease in the size of the aortic lumen. Intravenous angiocardiography is the safest and most practical method of study.

Fifteen cases are presented, 12 of which had angiocardiographic investigation.

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ADDENDUM

Since the preparation of this paper, 8 additional cases of dissecting aneuryms of the thoracic aorta have been studied by means of angiocardiography.

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SOME PROBLEMS IN THE ANGIOGRAPHIC DIAGNOSIS OF DISSECTING ANEURYSMS OF THE THORACIC AORTA*

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THE diagnosis of dissecting aneurysms of the thoracic aorta by angiographic methods is discussed in the preceding article,[1] based on a series of 15 cases which were observed during a 24 month period. In this article, an additional 5 cases are reported which were studied during the last 9 months and in which intravenous aortograms of the thoracic aorta presented certain problems in diagnosis. Comments on these problems will be made.

Intravenous aortography[2,3] performed in a patient with a dissecting aneurysm of the thoracic aorta reveals one or several findings. Occasionally, irregular narrowing of the main aortic channel is demonstrated. The most frequent finding, however, is widening of the wall of the aorta. Less commonly, the false channel is opacified by the contrast agent. This false channel is usually separated from the true aortic lumen by a thin translucent linear shadow caused by a part of the aortic wall separating the true lumen from the dissecting channel. Roentgenographic findings somewhat similar to these, but apparently not representing dissecting aneurysms, were encountered in the following cases.

REPORT OF CASES

CASE I. A 49 year old white woman presented at the hospital with sudden deep pain under her left breast. The pain radiated to the left lumbar area. The blood pressure was 150/80 in both arms. The patient had long fingers and a high arched palate. A family history of one parent dying of a ruptured aorta was reported by the patient. A chest roentgenogram was normal. An intravenous aortogram (Fig. 1) was made and was interpreted as showing a dissection beginning in the transverse portion of the arch of the aorta. At operation, the aorta was opened and explored thoroughly. No dissection was seen. The patient is alive and asymptomatic 18 months following surgical intervention.

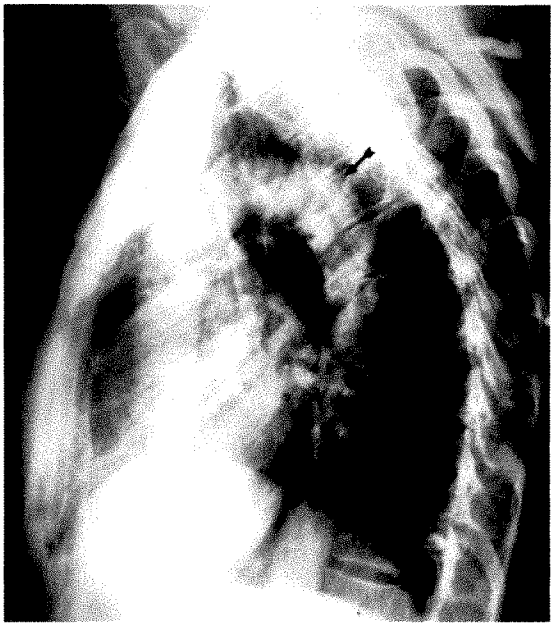


FIG. 1. Case 1. Intravenous aortogram in oblique lateral projection. The arrow indicates the area where contrast material was thought to be outside the lumen of the aorta.

CASE II. A 63 year old Negro female was admitted to the hospital because of severe substernal pain of 5 hours' duration. Many bouts of chronic congestive failure due to arteriosclerotic heart disease and associated hypertension preceded the present admission. She had been receiving digitalis and nitroglycerin for approximately 6 months prior to the present incident. The chest pain was substernal, crushing in type, and radiated into the right arm.

On physical examination the pulse was 80 and regular. The respirations were 36 per minute and the blood pressure was 180/80. The neck

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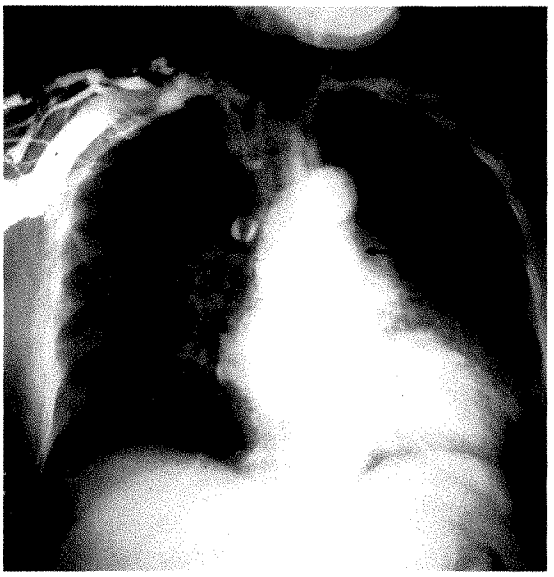


FIG. 2. Case II. Intravenous aortogram in frontal projection. The lumen of the descending aorta seems to be normal, but contrast material appears to be outside the aortic lumen, separated by a thin strip of radiolucency (arrow).

veins were distended and a blowing systolic murmur was heard along the left sternal border. The liver extended to a level 3 fingerbreadths below the right costal margin.

The chest roentgenogram showed evidence of generalized cardiomegaly with prominence of the left ventricle. The aorta was tortuous and dilated and the lung fields revealed early congestive changes.

An electrocardiogram showed left bundle branch block.

Because of the inability to document a myocardial infarction either by electrocardiography or clinical means and in view of the persistence of intermittent substernal chest pain, an intravenous aortogram was made to determine the exact configuration of the aorta and to exclude the remote possibility of a dissecting aneurysm (Fig. 2).

Approximately a month and a half after admission, the patient made slow progressive improvement and was subsequently discharged with the diagnosis of hypertensive arteriosclerotic heart disease associated with angina and mild congestive failure. The patient has been admitted to the hospital 3 months after this discharge for acute congestive failure and has subsequently been discharged.

CASE III. A 62 year old Negro man entered the hospital because of progressive dysphagia of 1 month's duration. Associated with this complaint was a 30 pound weight loss.

On physical examination, the blood pressure was 116/72. A suggestion of a pericardial friction rub was heard over the entire pericardium.

Since the chest roentgenogram revealed marked elongation and dilatation of the thoracic aorta, an intravenous angiocardiogram was made in order to exclude an aneurysm (Fig. 3). There was no evidence of an aneurysm, although the thoracic aorta was dilated.

The patient died 5 months after admission of carcinoma of the esophagus with mediastinal spread.

CASE IV. A 57 year old Negro male entered the hospital because of pain in the left arm and left flank of 6 hours' duration. The patient was well until 10 days prior to admission when he noted the onset of intermittent anterior chest pain.

Physical examination revealed the blood pressure to be 170/60. The pulse was 90 and respirations were 27 per minute. A systolic murmur was present to the left of the sternal border and radiated to the cardiac apex. Laboratory data were normal.



FIG. 3. Case III. Intravenous aortogram in the frontal projection. As in Figure 2 (Case II), contrast material appears to be outside the lumen of the descending thoracic aorta.

A routine chest study revealed a tortuous elongated aorta with the suggestion of an aneurysm of the aortic arch. An intravenous aortogram (Fig. 4) was made. Subsequent study of the patient revealed mild diabetes mellitus, controlled by diet alone. The patient was discharged from the hospital shortly thereafter. There has been no recurrence of chest pain since discharge.

Case v. A 54 year old longshoreman was admitted to the hospital with a 4 day history of fever and chills. A chest roentgenogram revealed a right lower lobe pneumonia. Microscopic hematuria also was found. Cystoscopy, and intravenous and retrograde pyelography were normal. The pneumonic infiltration, microscopic hematuria and fever cleared with antibiotic therapy. Because the patient's body measurements suggested a possible Marfan's syndrome and because the chest roentgenogram showed a large ascending aorta, possibly aneurysmal, intravenous aortography was performed. The lateral study (Fig. 5) showed what appeared to be a double lumen in the arch of the aorta. Further study, however, indicated that the "false channel" opacified earlier than the true aortic lumen (Fig. 6). It was suspected that this structure represented a vein, unusually dilated, possibly secondary to venous obstruction. Subsequent angiocardiograms proved that this was the situation (Fig. 7 and 8). Occlusion of the innominate vein was demonstrated. An extensive venous anastomotic system had developed. One of these dilated veins was the highest left intercostal vein, the course of which paralleled the transverse and descending portions of the aortic arch. Opacification of this vein, in a lateral projection, had resulted in a false "false channel."

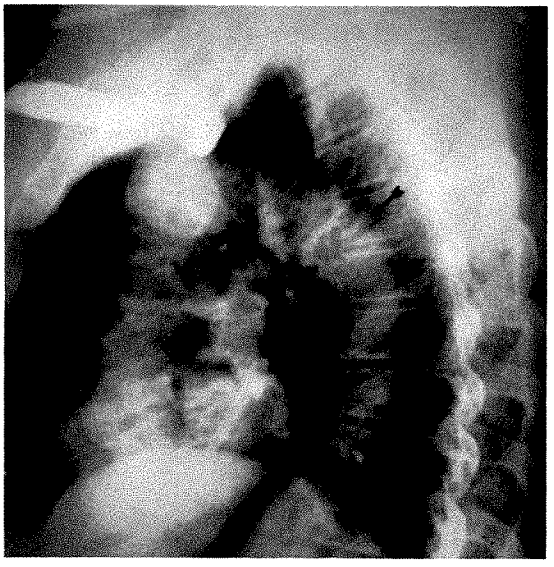


Fig. 4. Case iv. Intravenous aortogram in lateral projection. Contrast material appears to be outside the aortic lumen along the arch of the aorta (arrow).

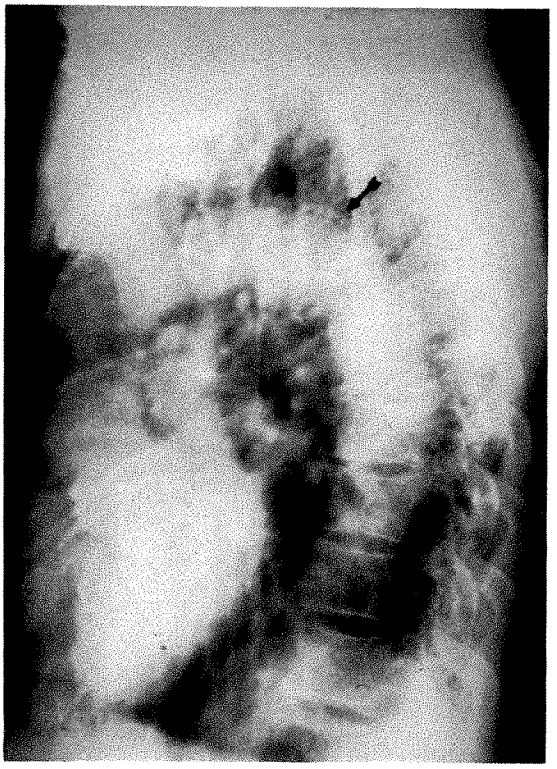


Fig. 5. Case v. Intravenous aortogram in lateral projection. The arrow points to what appears to be a "double lumen" of the arch of the aorta.

DISCUSSION

Our experience with the angiocardiographic demonstration of dissecting aneurysms of the thoracic aorta has indicated that the diagnosis can be made with certainty when the width of the aortic wall is shown to be more than 4 to 5 mm. In cases where contrast material is thought to be outside the lumen of the aorta but where the thickness of the aortic wall and/or the suspected false channel measures less

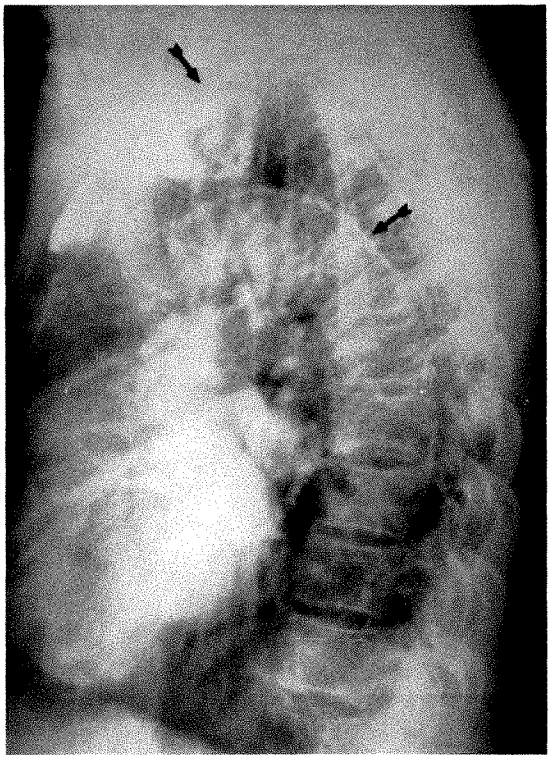


FIG. 6. Case v. An earlier roentgenogram in the series indicates that the "false channel" opacified earlier than the true aortic lumen.

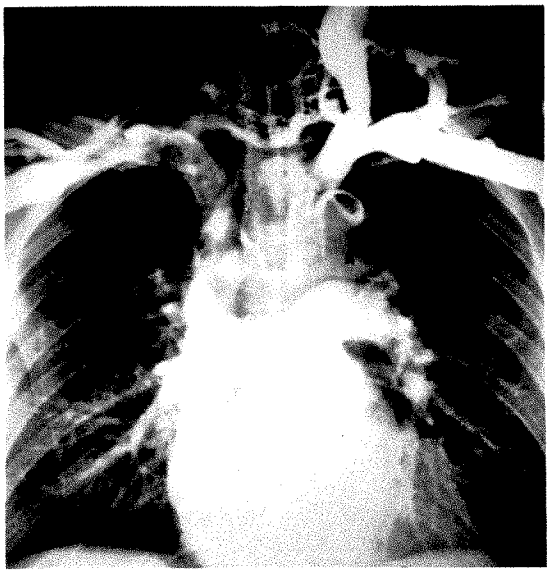


FIG. 7. Case v. Subsequent frontal angiocardiogram showed occlusion of the innominate vein. An extensive venous anastomotic system has developed. The arrow points to the dilated highest left intercostal vein.

than 4 mm. in width, caution in making the diagnosis is indicated.

Case I was examined early in our experience with the aortographic demonstration of dissections. The clinical evidence of a dissecting aneurysm was strong. The patient was thought to have the Marfan syndrome and she had experienced the sudden onset of severe chest pain. On the aortogram (Fig. 1) contrast material was thought to be outside the lumen of the aorta. Operative intervention was undertaken and a thorough surgical exploration failed to disclose a dissection. In fact, despite the apparent presence of contrast material outside the aortic lumen, the visualized aortic wall measured only 3 mm. in thickness. It is worthy of note that this finding was encountered only in the oblique projection, the frontal view being within normal limits.

Cases II, III, and IV were less problemat-

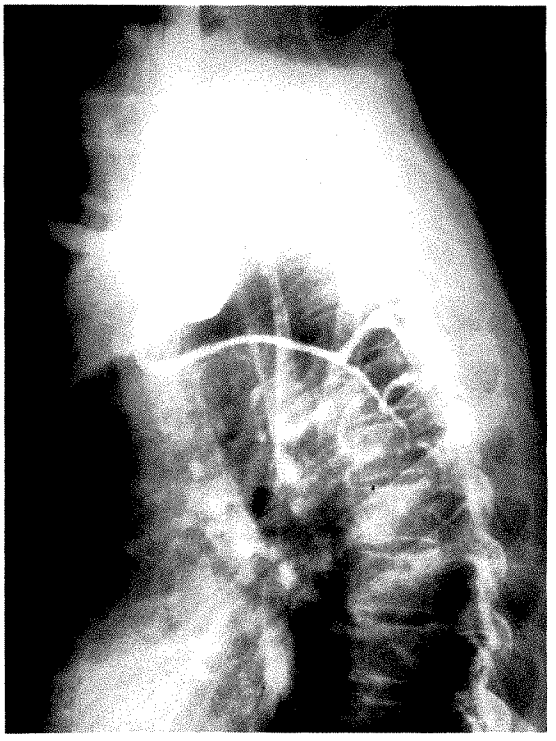


FIG. 8. Case v. The lateral angiocardiogram shows the course of the dilated highest left intercostal vein along the arch of the aorta. Opacification of this vein in the lateral aortogram had resulted in a false "false channel."

ical. The diagnosis of dissecting aneurysm was not suggested by the clinical history and aortographies were performed for other reasons. But again the studies suggested that, although the aortic channel was normal in appearance, contrast material appeared to be outside the aortic lumen, separated by a thin strip of radiolucency. In 2 of these cases, the finding was noted in the frontal projection; in the third instance, in the oblique lateral projection as in Case 1.

No definite explanation for this unusual opacification of what is thought to be a normal aorta is readily available. It is possible that opacification of adventitial vascular structures may be involved, and that contrast material in these vessels may be identified outside the aortic lumen and thus imitate a dissecting aneurysm. In other instances, it is possible that opacification of venous structures such as the highest intercostal vein in the region of the aortic arch may simulate a dissection of the thoracic aorta. In Case V, the highest left intercostal vein, dilated secondary to obstruction of the innominate vein, followed the course of the aortic arch. When both the vein and the aorta were opacified, a false "false channel" could be misinterpreted on the late serial roentgenograms.

The possibility that an elongated tumor mass contiguous to the aorta might resemble a thickened aortic wall suggested itself.[4] However, the appearance of the aortic lumen and wall on multiple projections might assist in differentiating this pathologic entity from a dissecting aneurysm.

SUMMARY AND CONCLUSIONS

Several cases illustrating certain problems and errors in the angiographic diagnosis of dissecting aneurysms of the thoracic aorta are presented. In these cases contrast material appeared to be outside the lumen of the aorta but the width of the aortic wall measured less than 4 to 5 mm. It is suggested that this opacification, simulating a dissection, may represent either adventitial vascular opacification or venous structures.

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ROENTGEN FEATURES OF THREATENING THORACIC AORTIC RUPTURE*

REPORT OF 4 CASES

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RECENTLY, in a discussion of the pathogenesis of impending thoracic aortic rupture, it was pointed out that the radiologist, because he was often the first to see the roentgenogram, had a unique role in diagnosing threatening aortic rupture.[3] Furthermore, because of improvements in surgical techniques, especially the development of a pump oxygenator, plastic grafting materials, and blood replacement, treatment by resection of even the ruptured aorta has become practical and, indeed, offers the only hope of survival.

The thoracic aorta, surrounded by air containing tissues in much of its course, is roentgenographically visible. Calcium in the aortic wall further enhances visibility when arteriosclerosis is present. However, when dilatation, tortuosity, unfolding, and kinking of the thoracic aorta occur, it may be difficult, if not impossible by conventional roentgenography to distinguish it from the surrounding structures. The recognition of thoracic aneurysm may also be rendered more difficult by an intraluminal thrombus preventing the usual pulsation. Contrast visualization of the thoracic aorta either via the intravenous route,[1] or by percutaneous retrograde femoral aortography, has provided safe and practical methods of visualizing the thoracic aorta.[2] When rupture of an aneurysm threatens, there are often premonitory symptoms and signs. Findings of conventional and contrast roentgenograms of the thorax also may permit diagnosis of impending aortic rupture. Herein are reported 4 cases illustrating the roentgen aspects of threatened aortic rupture.

REPORT OF CASES

CASE 1. A 65 year old Caucasian housewife (N.Y.H. No. 455268) was admitted for the fourth time in April, 1959, with a complaint of chest pain of 8 days' duration. She had been first admitted in 1949, because of failing vision. She had a history of having had a positive serologic blood test (Wassermann) in 1932. Advanced primary pigmentary degeneration of the retina of both eyes was the only abnormal finding on physical examination. The spinal fluid revealed a normal serologic test (Wassermann) with a normal colloidal gold curve. After a 10 day course of penicillin therapy, she was discharged. The second admission was in 1956, for pain in the left chest. Her blood pressure was 180/70 mm. Hg, and her heart was enlarged to the left of the anterior axillary line. Diastolic and systolic murmurs were heard at the base of the heart. The electrocardiogram was normal, and she was again treated with penicillin. Later, in 1956, she was re-admitted because of precordial pain radiating to the left arm. The blood pressure was 218/78 mm. Hg, and the heart was enlarged. Continuous systolic and diastolic murmurs radiating into the neck were again heard in the aortic area. A roentgenogram of the chest showed enlargement of the left ventricle with prominence of the aortic knob. A 2 cm. aneurysm was detected lateral to the calcium-defined border of the aortic arch (Fig. 1*A*). There were also calcifications of the ascending aorta.

Eight days prior to the final admission (April, 1959), the patient developed sudden onset of dyspnea, palpitation, vertigo, nausea,

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† Recipient of investigatorship of the Health Research Council of The City of New York under grant I-258.

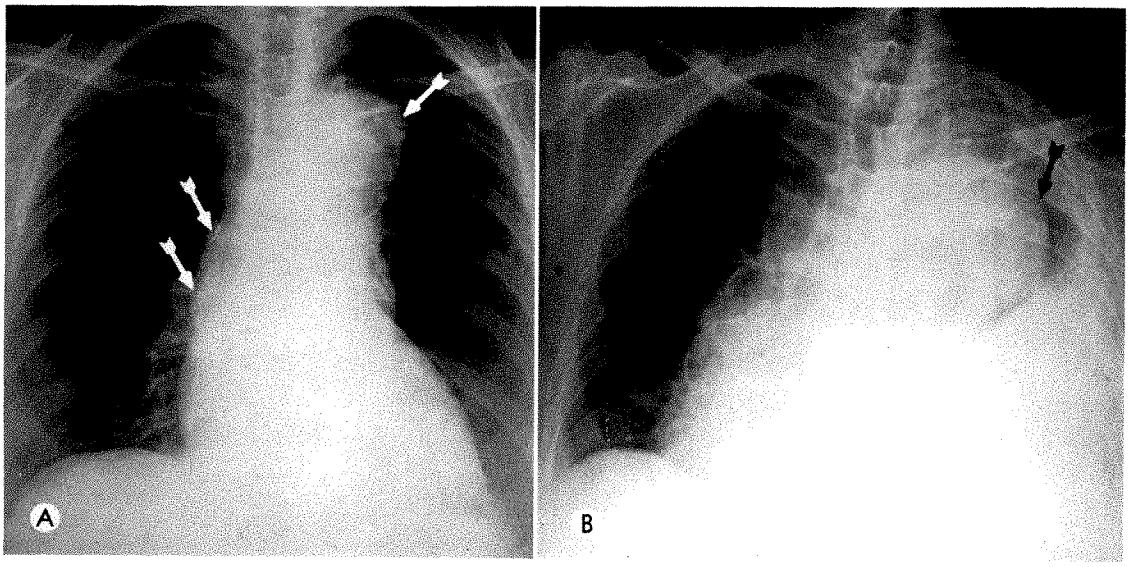


Fig. 1. Case 1. *Aortic rupture into the left pleural space.* (*A*) Teleroentgenogram made 3 years prior to rupture of an aortic aneurysm (upper arrow). Note the enlarged left ventricle and dilated ascending aorta, lined with calcium (lower arrows). (*B*) Bedside roentgenogram of the chest showing the huge aortic aneurysm (arrow) and left pleural effusion displacing the heart.

vomiting, and severe pain and tenderness in the anterior and lateral aspects of the left chest radiating into the left arm.

Physical examination disclosed an obese, pale woman in respiratory distress, breathing rapidly. The pulse was also rapid and the blood pressure was 170/70 mm. Hg. The trachea was shifted to the right and signs of fluid were elicited in the left hemithorax; the right lung was clear. The heart sounds were distant, the rhythm was regular, and a loud systolic murmur was heard at the base of the heart. The pulse was Corrigan in type.

Laboratory data revealed: hemoglobin, 12 gm./100 cc.; erythrocytes, 3.6 million/cu.mm.; leukocyte count, 12,800/cu.mm.; and sedimentation rate (Wintrobe), 43 mm. in 1 hour. The blood serologic test (Mazzini) was doubtful, and the Wassermann test was negative. A roentgenogram showed a markedly enlarged aortic shadow with cardiac displacement due to a huge left pleural effusion (Fig. 1*B*). Rupture of an aneurysm of the aorta was diagnosed and confirmed by thoracentesis of grossly bloody fluid. The surgical consultant was reluctant to attempt the repair of the aortic rupture in view of her long standing aortic valvular regurgitation. The patient became more dyspneic, fluid reaccumulated in the left hemithorax, and finally coma and death followed 8 days after admission and 16 days after the onset of symptoms.

Postmortem examination revealed 2,000 cc. of dark, bloody fluid in the left pleural cavity from a ruptured aortic aneurysm. The left lung was almost completely collapsed and showed atelectasis. The heart weighed 520 gm.; the left ventricular wall measured 23 mm., the right was 7 mm. The aortic valve was moderately insufficient, but the aortic cusps and commissures were intact, translucent, and normal. The coronary arteries were markedly thickened, atherosclerotic, and the lumina of the branches of the anterior descending and circumflex arteries were pinpoint in size with scarring of the myocardium in their distribution. Extensive interstitial myocardial fibrosis was seen. The thoracic aorta was markedly dilated, thin, and extensively involved with yellow, friable, atheromatous plaques. Approximately 1.5 cm. beyond the origin of the left subclavian artery on the convexity of the arch, there was a saccular aortic aneurysm measuring 7 cm. in greatest diameter and partially filled with old and recent laminated thrombus. In the lateral inferior margin of the aneurysm, there was a 1 cm. rupture. Extensive arteriosclerosis of the abdominal aorta and its greater branches was also

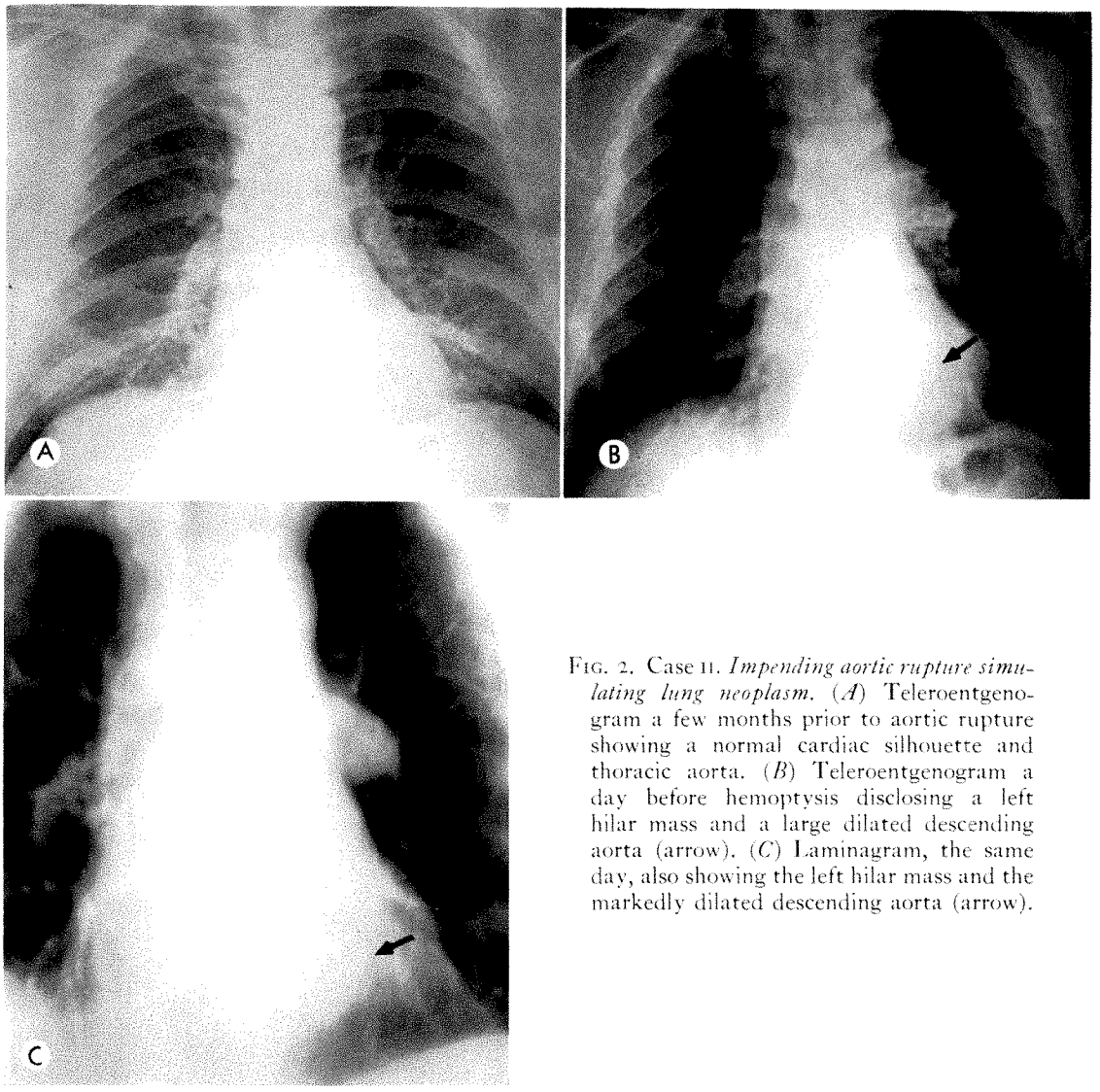


FIG. 2. Case II. *Impending aortic rupture simulating lung neoplasm.* (*A*) Teleroentgenogram a few months prior to aortic rupture showing a normal cardiac silhouette and thoracic aorta. (*B*) Teleroentgenogram a day before hemoptysis disclosing a left hilar mass and a large dilated descending aorta (arrow). (*C*) Laminagram, the same day, also showing the left hilar mass and the markedly dilated descending aorta (arrow).

present. The final anatomic diagnosis was syphilitic cardiovascular disease with aortitis, aortic insufficiency, and ruptured aortic aneurysm into the left pleural cavity.

CASE II. A 50 year old man had always been well except for intermittent pain over the neck of several years' duration. A roentgenogram of the chest (August 28, 1959) disclosed a left hilar mass not present previously. He was hospitalized elsewhere. Physical examination was normal. Roentgenograms and laminagrams revealed a small left hilar mass and increased convexity of the descending aorta (Fig. 2, *A*, *B* and *C*). The electrocardiogram was normal. Bronchoscopy showed the left main bronchus displaced medially by an extrinsic mass. Aspirated exudate revealed no abnormal cells. Angiocardiograms were erroneously interpreted as showing a nonvascular left hilar mass. The patient had a slight fever, blood streaked sputum, leukocytosis, and increasing back pain for the next 9 days. Suddenly, he became dyspneic, cyanotic, coughed up a large blood clot and died.

Postmortem examination revealed a massive (2,500 cc.) clotted hemorrhage in the left pleural space caused by a rupture of the thoracic aorta. There was an area of hemorrhage 6 cm. in diameter in the left hilus. This had dissected beneath the pleura and into lung parenchyma. The heart showed only mild coronary artery

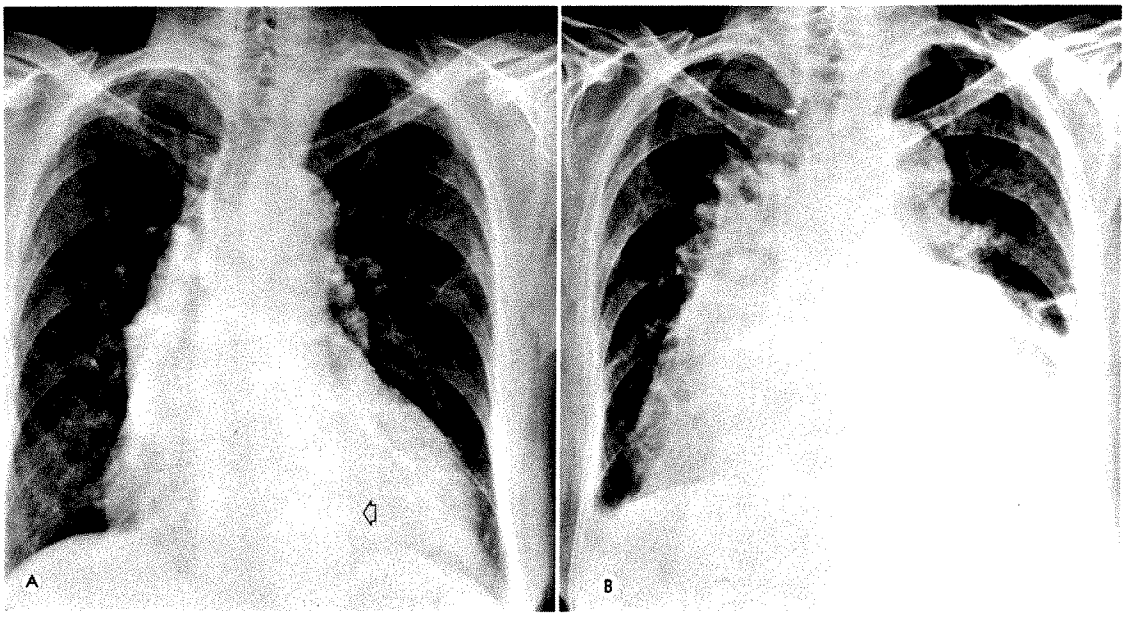


Fig. 3. Case III. *Rupture of aorta into pericardium.* (*A*) Chest roentgenogram made on the first day of admission shows the heart to be enlarged and the descending aortic shadow is visible through the cardiac silhouette (arrow). (*B*) Roentgenogram made on the third hospital day discloses marked increase in the cardiac silhouette. Pleural fluid is present bilaterally and the aortic knob is enlarged. The rapidity of cardiac enlargement (judged by intervening roentgenograms) and the configuration of the heart on this and other roentgenograms indicate a hemopericardium.

calcification. The aorta was tortuous and atheromatous, especially the descending and abdominal portions. The aorta was dissected from the region of the third to the tenth thoracic vertebrae. Below, in the region of the tenth thoracic vertebra, there was a saccular 4 by 3 by 3 cm. nonruptured dilatation of the descending aorta. Another 3 by 3 by 3 cm. (intact) aneurysm was present at the bifurcation of the abdominal aorta. Death had resulted from massive rupture of a slowly leaking dissecting aneurysm of the thoracic aorta into the pleural cavity.

Case III. Two days before hospitalization, a 75 year old housewife (U.O.M.S. No. 2-12-07) experienced the abrupt onset of profound generalized weakness and severe precordial pain which radiated into the right upper abdomen. Physical examination revealed dullness at the lung base, soft apical systolic and rough aortic diastolic murmurs. Her record indicated seropositive syphilis. Chest roentgenograms made on the first and third hospital days (Fig. 3, *A* and *B*) showed a marked interval increase in the size of the cardiac silhouette and bilateral pleural fluid. During the remaining 3 days of her life, she complained of a smothering sensation whenever she tried to lie down. Death came suddenly and was ascribed to congestive heart failure.

At autopsy, the aorta was found to be ruptured. Approximately 1,000 cc. of blood had entered the pericardial sac through a 1 cm. tear a short distance above the posterior aortic valve cusp. In addition, there was diffuse, probably acute hemorrhage extending into pulmonary interstitial tissue along the pulmonary arteries and interlobar fissures. It was estimated that blood had been in the pericardial space for at least 2 or 3 days, and probably accounting for the duration of symptoms. While the aorta exhibited severe arteriosclerotic change, it was not significantly dilated, and did not present the usual finding of syphilitic aortitis.

Case IV. A 12 year old boy (U.O.M.S. No. 26-20-69) was admitted to the hospital because of a sharply delineated, ovoid density in the left perihilar region which appeared 4 months following the apparently successful resection and end-to-end re-anastomosis of a classic coarctation of the aorta. Although the mass was nonpulsatile, its shadow merged with that of the

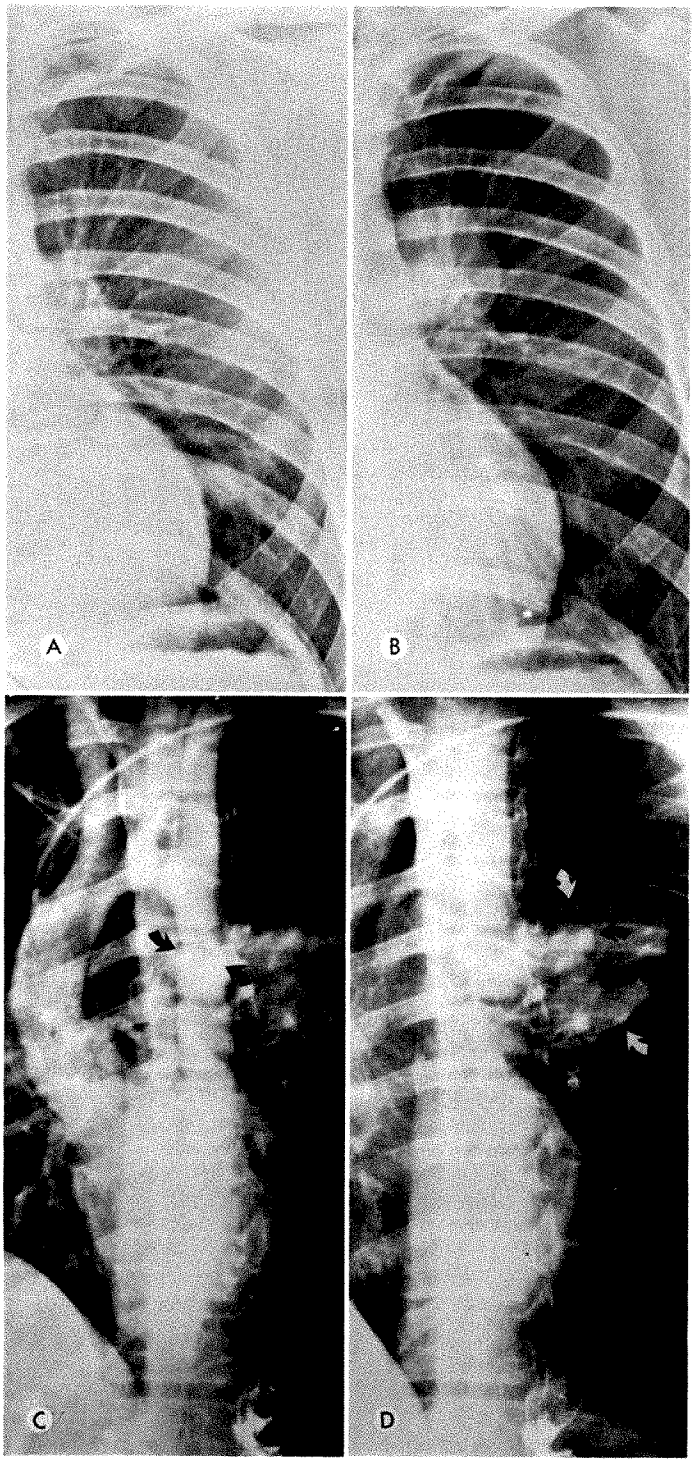


FIG. 4. Case IV. *Aneurysm following resection of coarctation of the thoracic aorta.* (*A*) Chest roentgenogram made 4 months prior to admission and 2 months after resection of the coarctation of the aorta. (*B*) One month before surgery, adjacent to the line of operative re-anastomosis, a gradually enlarging, rounded density projects into the left lung field. (*C*) Left anterior oblique angiocardiogram shows a normal left ventricle and ascending aorta. At the site of previous operative repair (black arrows) the aortic lumen is

aorta and appeared to be progressively enlarging (Fig. 4, *A* and *B*). Emergency angiocardiography (Fig. 4, *C* and *D*) revealed that the left hilar density was a thick walled false aneurysm arising and filling from the descending aorta at the level of the previous aortic anastomosis. At subsequent thoracotomy, the diagnosis was confirmed, the lesion resected, and a graft inserted in the resulting gap. The patient is alive and well. Examination of the surgical specimen indicated that slow leakage at the suture line had caused formation of a false aneurysmal cavity within an envelope of reactive inflammatory connective tissue. Layered thrombus added to the thickness of the wall.

DISCUSSION

In a recent paper,[3] it was emphasized that diagnosis of impending rupture of the aorta depended on recognition of transmural hemorrhage and aortic enlargement. Both of these factors were present in the 4 cases described above. In Case I, diagnosis of aortic rupture became evident following sudden chest pain and left pleural effusion in the 65 year old syphilitic woman who had had an enlarging aortic aneurysm (Fig. 1*B*). Thoracentesis established the diagnosis of rupture of the aorta 9 days before death. Unfortunately, operation was not attempted. In Case II, practically all the signs and symptoms of threatening aortic rupture went unrecognized during the 9 days preceding death. These included a left hilar mass, marked convexity of the descending aorta which had not been present in a previous roentgenogram of the chest (Fig. 2, *A* and *B*), and hemoptysis. In retropsect, the angiocardiogram showed opacification of a left hilar mass, but the presumptive diagnosis of lung cancer was uppermost and may have led to the incorrect interpretation of a nonvascular mass. In Case III, the contour as well as the magnitude and rapidity of enlargement of the cardiovascular silhouette gave presumptive roentgenologic evidence of a hemopericardium (Fig. 3*B*). Lacking clinical or laboratory proof of myocardial infarction or an inflammatory process, a mechanical explanation (pericardial tamponade), and perhaps an effort at surgical correction would have been justified. In Case IV, the patient's recent history of repair of a coarctation and roentgen evidence of an enlarging para-aortic mass (Fig. 4, *A* and *B*) led to the establishment by contrast visualization (Fig. 4, *C* and *D*) of the correct diagnosis, and subsequent lifesaving surgery.

SUMMARY AND CONCLUSIONS

Thoracic aortic rupture was fatal in 3 of the 4 patients. In the first case, progressive enlargement of an aortic aneurysm was observed for several months in a syphilitic woman with aortic regurgitation. Aortic rupture began with severe chest pain and hemothorax. Despite the rupture, the patient lived for 16 days. Even though postmortem study revealed extensive coronary artery and aortic atherosclerosis, resection of the aneurysm and replacement with a graft might have prolonged life. In the second case, failure to recognize the significance of hemoptysis, mediastinal hemorrhagic seepage, and progressive dilatation of the descending aorta resulted in a mistaken diagnosis of lung cancer. Here too, 9 days elapsed before fatal left hemothorax ensued. In the third patient, failure to appreciate the significance of a rapidly enlarging cardiac silhouette prevented the

poorly shown. (*D*) Three seconds later, the periphery of the para-aortic mass (between white arrows) is nonvascular, and a 1 by 2 cm. ovoid central cavity (black arrow) fills from the aorta at the site of surgery. Delayed emptying is shown. This is the characteristic appearance of a false aneurysm at an aortic suture line. Laminated thrombus and reactive change account for the thickened wall.

diagnosis of hemopericardium due to localized aortic rupture just above the posterior aortic cusp. In this instance, cardiac tamponade allowed the patient to live only 5 days. In the last case, progressive enlargement of the descending aortic shadow 4 months after resection for coarctation of the aorta was demonstrated to be a thick walled false aneurysm by angiocardiography. Resection of the aneurysm and insertion of a graft were curative.

Fatal rupture of the aorta or thoracic aortic aneurysm, including those classified as dissecting, is often preceded by transmural seepage of blood and progressive dilatation of the aorta at the site of impending rupture. Premonitory manifestations may precede catastrophic rupture by several weeks or only a few hours, but may provide ample opportunity to establish the diagnosis in time to prevent death. Clinical findings of importance include those of unexplained hemoptysis, a rapidly expanding mediastinal mass, the sudden appearance and rapid accumulation of blood in the pleural or pericardial spaces, known disease, or predisposition to disease of the aorta, and the occurrence of severe chest pain unexplained by electrocardiograms. A diagnosis of acute dissecting aneurysm implies probably, though not inevitably, death through external rupture.

The significant manifestations of thoracic aortic rupture include progressive enlargement and loss in border sharpness of the aorta or an aortic aneurysm, especially when accompanied by adjacent pulmonary infiltrative densities and of the sudden appearance and rapid accumulation of pleural or pericardial fluid; particularly in the presence of collateral evidence of aortic disease. Pitfalls include bronchogenic carcinoma, bronchopneumonia, mediastinal or hilar lymph node enlargement, and heart failure. The most common error is failure to recognize the presence or significance of interval changes in the aortic margin. Once suspected, the diagnosis of impending aortic rupture can be confirmed decisively by contrast visualization, the risk of which is negligible by comparison to that of the missed diagnosis.

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VOL. 91, No. 6

TRAUMATIC ANEURYSM OF THE THORACIC AORTA*

A FURTHER REPORT

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IN 1958, the roentgen features of acute and chronic types of traumatic aneurysm of the thoracic aorta were published in this JOURNAL.[18] Since then, 3 patients with chronic traumatic aortic aneurysms were studied at this center. In addition, the patient reported by Sarot and Lazzarini[14] in 1955 had angiocardiographic visualization of the end-to-side homograft which had been inserted 9 years previously following a traumatic aneurysm of the thoracic aorta that had occurred in an automobile accident 14 years earlier. These data, with a brief review of some of the literature on traumatic aneurysms of the thoracic aorta published since 1957,[1,2,5–13,15,16] form the basis of this report and serve to re-emphasize the importance of having a high index of suspicion whenever the characteristic location of a traumatic aneurysm, at the top of the thoracic aorta, is encountered.

REPORT OF CASES

CASE 1. An asymptomatic 45 year old man (N.Y.H. No. 926510) was referred following the finding of a calcified mass in the mediastinum after a routine roentgenogram of the chest (Fig. 1*A*). At the age of 12 years in an automobile accident he struck his chest against the dashboard. He was hospitalized for a week and was treated by adhesive strapping of the thorax. Following this, he was well and rarely visited a

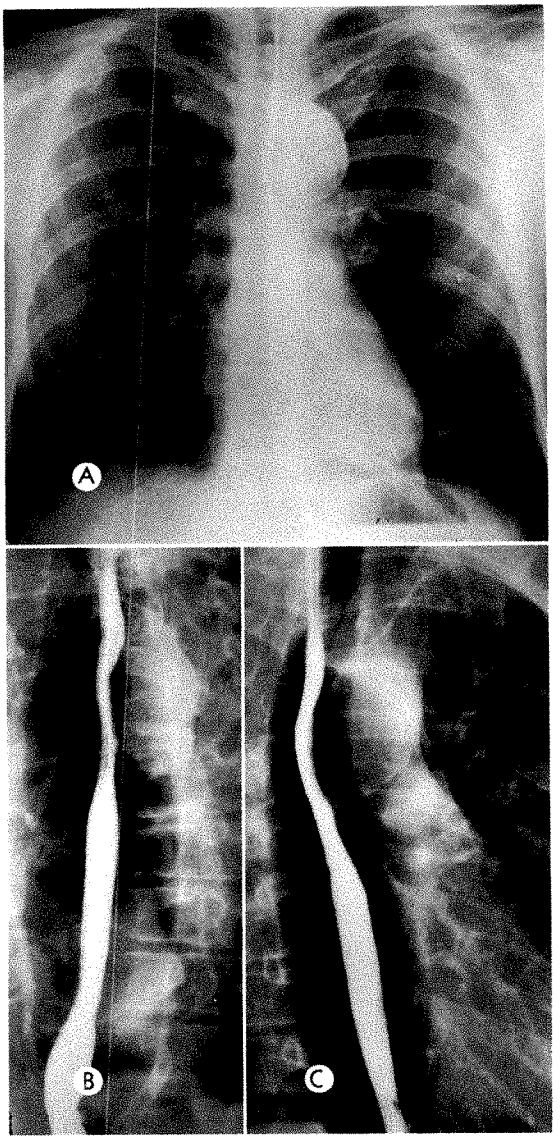


FIG. 1. Case 1. *Chronic traumatic aneurysm of the thoracic aorta of 33 years' duration.* (*A*) Frontal esophagram showing a large left superior mediastinal mass lined with calcium in the region of the aortic knob. (*B*) Left anterior oblique esophagram demonstrating the mass behind the esophagus and located at the top of the descending aorta. (*C*) Right anterior oblique esophagram disclosing the large calcium rimmed mass indenting the esophagus.

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† Recipient of Investigatorship of the Health Research Council of the City of New York under contract I-258.

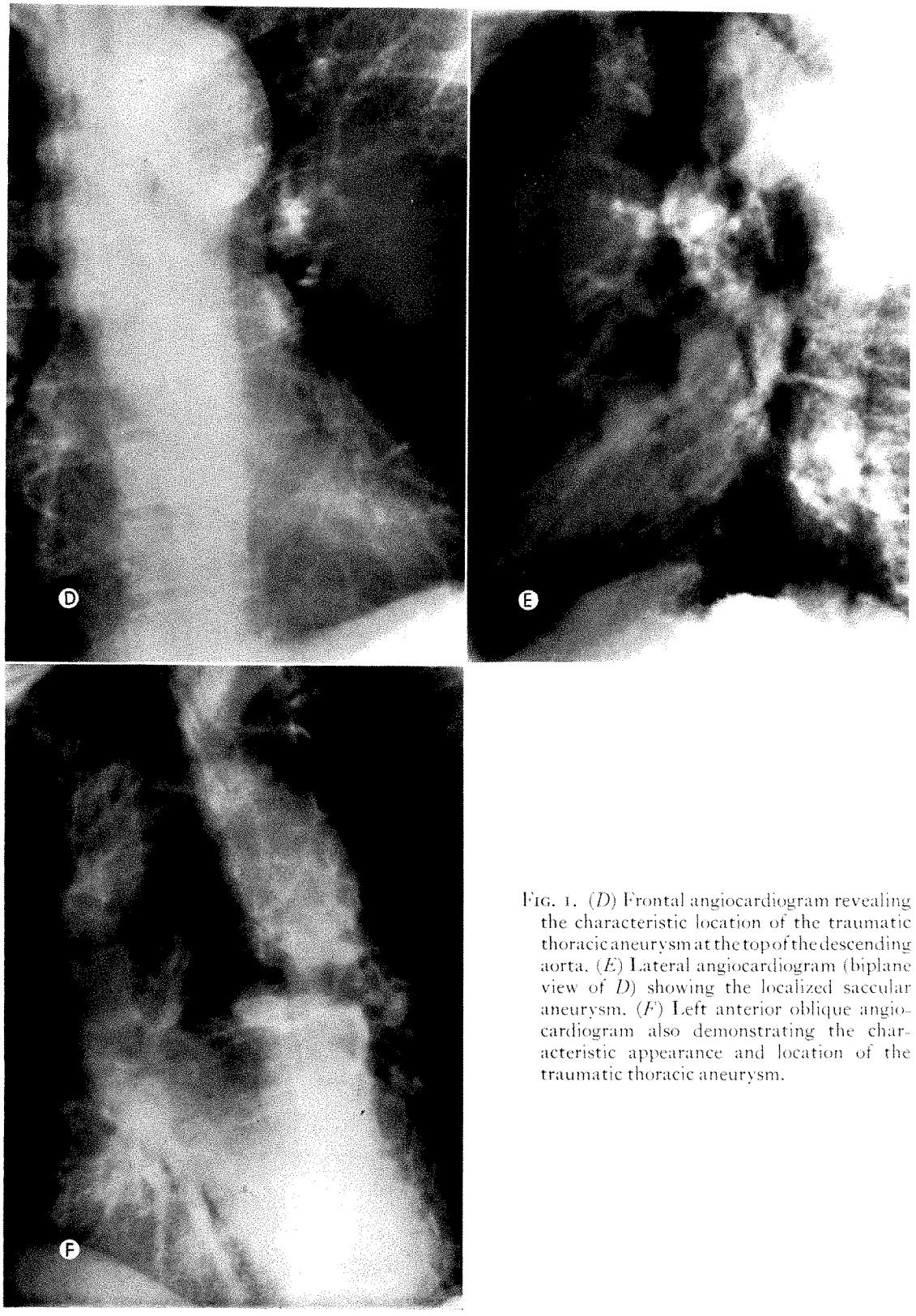


FIG. 1. (*D*) Frontal angiocardiogram revealing the characteristic location of the traumatic thoracic aneurysm at the top of the descending aorta. (*E*) Lateral angiocardiogram (biplane view of *D*) showing the localized saccular aneurysm. (*F*) Left anterior oblique angiocardiogram also demonstrating the characteristic appearance and location of the traumatic thoracic aneurysm.

physician. Recently, because of a cold, a roentgenogram of the chest was ordered.

Physical examination was entirely normal. The blood pressure was equal in each arm, measuring 140/90 mm. Hg. Serologic studies for syphilis were negative; he also denied venereal disease. Conventional roentgenography of the chest disclosed a large linear calcified left superior mediastinal mass in the region of the aortic knob which indented the esophagus medially (Fig. 1*A*) and posteriorly (Fig. 1, *B* and *C*). Biplane simultaneous angiocardiograms on October 24, 1962, showed a 7 by 5 cm. saccular aneurysm containing linear calcifications, beginning just beyond the origin of the left subclavian artery (Fig. 1, *D* and *E*). Another angiocardiographic study, in the left anterior oblique view, also demonstrated the aneurysm at the top of the descending aorta (Fig. 1*F*). Resection of the aneurysm and replacement by a synthetic material graft was advised, especially because of the good condition of the patient. As yet surgery has not been accepted by the patient.

Case II. A 28 year old male pedestrian was struck by a car in 1950. He had been flung to the ground and had sustained fractures of the right leg and injuries to the head. In 1957, an abnormal shadow was discovered on a routine roentgenogram of the chest. In 1962, a left chest operation was performed. While on his honeymoon in New York in 1963, another routine chest roentgenogram was made. It demonstrated a large left mediastinal mass lined with calcium in the characteristic location of a traumatic thoracic aneurysm, at the top of the aorta (Fig. 2). The report of the findings upset the patient very much because he had been led to believe that the thoracic lesion had been removed by the operation. He was informed that the roentgenogram showed evidence of a left thoracotomy (Fig. 2), but that the mediastinal lesion had apparently not been resected. The patient promised to return for further studies and surgical excision of the aneurysm.

Case III. A 33 year old man (N.Y.H. No. 852582) fell from the window of his ninth floor apartment to the ground and sustained multiple compound fractures of the legs, 9 years prior to admission to the hospital. Osteomyelitis of the left leg developed. In the ensuing years, he had had several surgical procedures for relief of this complication. During an admission to a neighboring orthopedic hospital in 1957, a routine

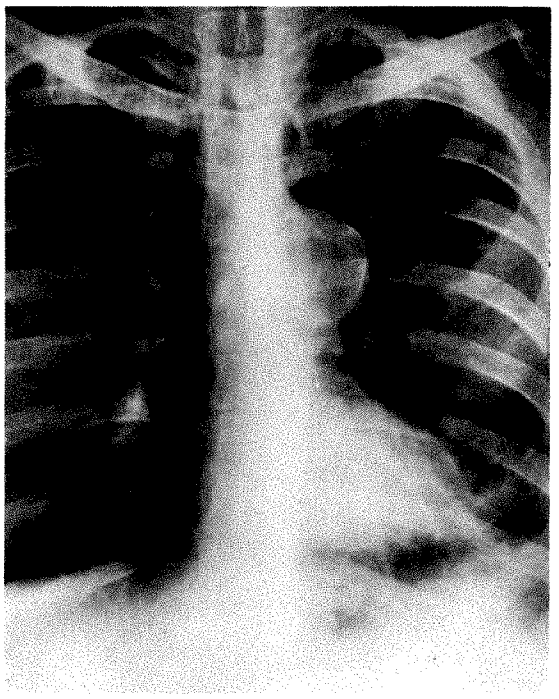


Fig. 2. Case II. *Chronic traumatic aneurysm of 7 years' duration.* Frontal teleroentgenogram of the chest showing a large calcified mass in the upper left hilus.

roentgenogram of the chest disclosed a large bulge in the region of the aortic knob. He denied having chest pain, hoarseness, hemoptysis, cough, chills, or fever.

In 1959, re-examination of the chest showed the left mediastinal mass to be somewhat larger (Fig. 3*A*). Angiocardiographic study on June 16, 1959, showed a large 4 by 4 cm. saccular aneurysm at the top of the aorta (Fig. 3, *B* and *C*). The blood pressure was equal in both arms and measured 140/90 mm. Hg. Roentgen studies of the lower extremities showed old healed fracture of the mid-portion of each femoral shaft, with an intramedullary rod in the right femur. Resection of the aneurysm and replacement by a synthetic graft were done on June 1, 1960. At gross examination the aneurysm measured 4 by 4 cm., the adventitia and intima were smooth and there was no rupture. Histologic section of the aneurysm showed a thin intima and hyalinized wall. Paralysis of the recurrent nerve followed the operation. Otherwise, recovery was uneventful.

Recent follow-up examination disclosed that the patient was in good health. The conventional roentgenogram of the chest showed a hypoplastic aortic arch (Fig. 3*D*). Simultaneous

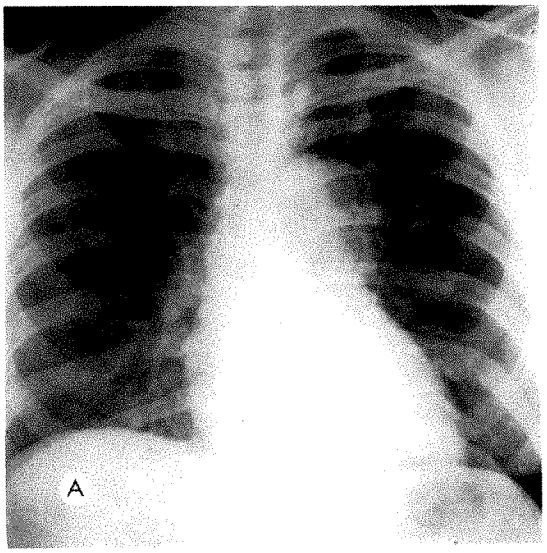


FIG. 3. Case III. *Chronic traumatic aneurysm of 9 years' duration; resection performed despite asymptomatic state of the patient because of enlargement of aneurysm on serial roentgen studies.* (*A*) Frontal teleroentgenogram of the chest showing a left upper mediastinal bulge without calcification in the region of the aortic knob.

biplane angiocardiographic studies, in frontal and lateral views, revealed a well functioning prosthetic graft in the thoracic aorta (Fig. 3, *E* and *F*).

CASE IV. In 1954, a 38 year old woman developed severe cough and left shoulder pain 14 years after a severe automobile accident. She consulted Dr. Irving A. Sarot, and he established that there was a traumatic thoracic aortic aneurysm (Fig. 4*A*).[14] At operation on October 20, 1954, the aneurysm was resected and aortic arch continuity was secured by an end-to-side homograft. The patient had been asymptomatic and well for the past 9 years (Fig. 4, *B* and *C*). In order to visualize the graft more clearly, angiocardiographic studies in frontal and left anterior oblique views were made on November 1, 1962. An intact and well functioning end-to-side thoracic aortic homograft was visualized (Fig. 4, *D* and *E*).

DISCUSSION

Rupture of the thoracic aorta due to blunt trauma occurs at the top of the aorta. This is explained by the physical forces which are concerned with deceleration acting with undue stress on the aorta, producing a tear just beyond the point of fixation by the brachiocephalic arteries and the insertion of the ligamentum arteriosum. In the majority of cases, the aortic rupture is through-and-through and involves all the layers of the aorta. In a small group of cases

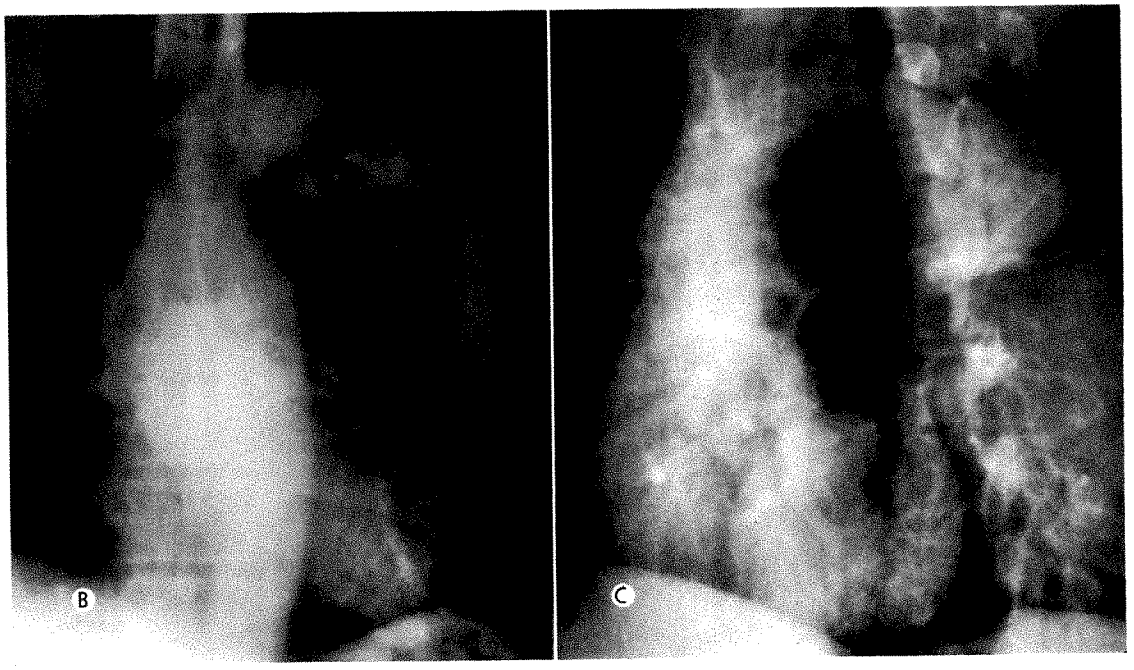


FIG. 3. (*B*) Frontal angiocardiogram revealing a saccular aneurysm at the top of the aorta. (*C*) Left anterior oblique angiocardiogram also demonstrating the aneurysm.

rupture is incomplete, the adventitia being preserved and dissected from the medial layer by thrombus. When all the layers of the thoracic aorta are torn, fatal hemorrhage usually results. In a few, especially when the adventitia is spared, there may be organization of the thrombus with a connective tissue wall and formation of a false aneurysm following recovery. In the chronic stage, deposition of calcium in the aneurysm very often takes place.

Traumatic aneurysms of the thoracic aorta may be classified as acute and chronic.[17,18] Following an automobile accident, unconsciousness, shock, multiple fractures, and internal injuries may divert attention away from the thorax, especially when the external evidence of thoracic injury is trivial, resulting in an overlooked rupture of the aorta. It is for this reason that routine serial roentgenograms of the chest are recommended for every patient involved in a vehicular accident; and this, with a high index of suspicion, has resulted in diagnosis of aortic rupture during life.[4,10,12,13,15,16,18] Early, following injury, serial roentgenograms may reveal mediastinal bleeding by showing progressive mediastinal widening, obscuration of the aortic arch, and fluid in the left hemithorax.[4] Later, in a few days to 3 or 4 weeks, a localized bulge appears adjacent to and indistinguishable from the aortic knob; the aneurysm then becomes chronic, localized, and distinct. Within a year, a large dense rounded mass appears at the top of the aorta—a characteristic location. With the passage of time, calcium may be deposited in the walls of the chronic traumatic aneurysm in layers or diffusely.[18]

Often, the finding of a mediastinal mass containing calcium in a chance or routine

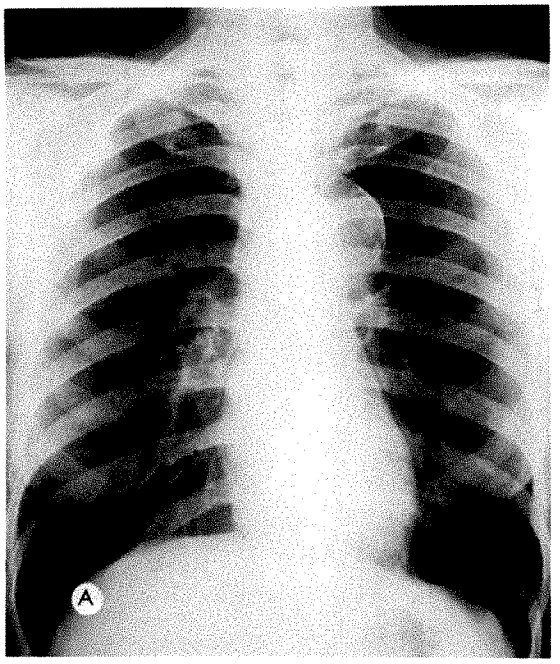


Fig. 4. Case IV. *Angiocardiographic demonstration of an intact end-to-side homograft made 9 years after resection of a chronic traumatic aneurysm of the thoracic aorta.* (*A*) Frontal teleroentgenogram of the chest made 14 years after an automobile accident because of severe cough and left shoulder pain.

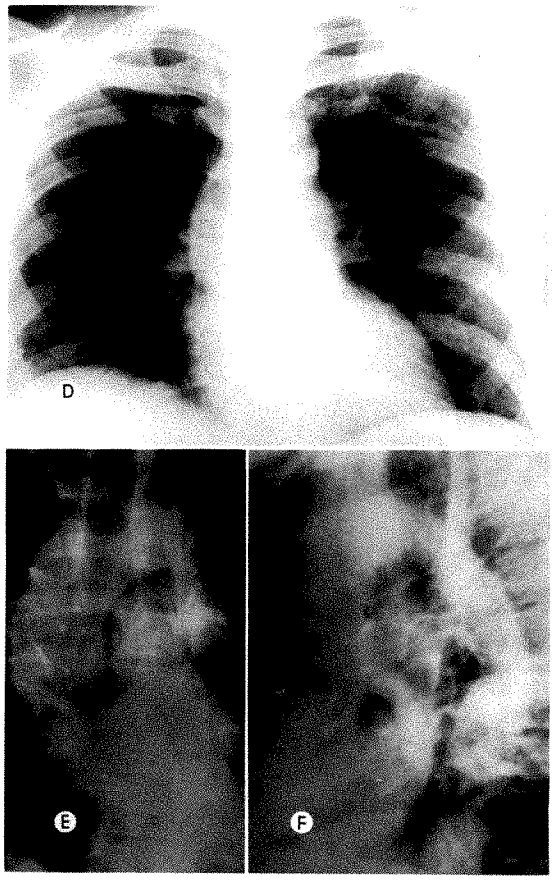


Fig. 3. (*D*) Frontal conventional roentgenogram of the chest made 3 years following resection of the traumatic aneurysm. (*E*) Frontal angiocardiogram revealing the intact teflon graft replacing the aneurysm. (*F*) Lateral angiocardiogram (biplane view of *E*) showing the intact graft to better advantage.

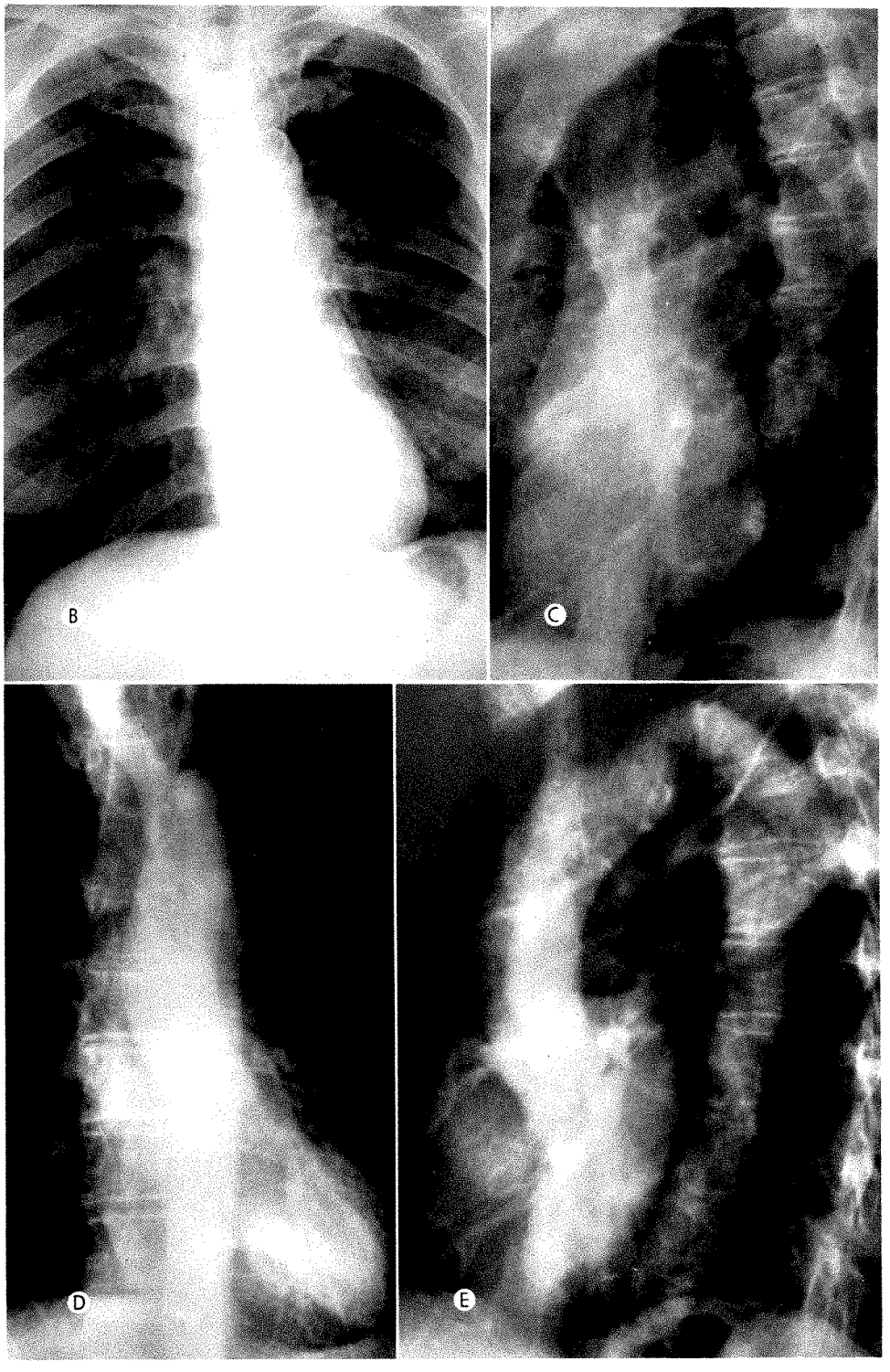


FIG. 4. (*B*) Conventional frontal teleroentgenogram of the chest made 9 years after resection of the chronic traumatic aneurysm of the thoracic aorta. Note the calcification of the wall of the graft. (*C*) Left anterior oblique roentgenogram of the chest also shows calcification of the homograft. (*D*) Frontal angiocardiogram showing the intact homograft. (*E*) Left anterior oblique angiocardiogram showing the end-to-side appearance of the homograft. (Figure 4*A* published through the courtesy of Dr. Irving A. Sarot.[14])

roentgenogram of the chest in an asymptomatic individual may be the first inkling of a chronic traumatic aneurysm. After angiocardiography demonstrates that the mediastinal mass is vascular, the characteristic location at the top of the aorta, the absence of a positive serologic test for syphilis, and the history of trauma establish the diagnosis.[11,18] In other patients chest pain, cough, hoarseness, or hemoptysis may lead to roentgen study of the thorax and discovery of a left superior mediastinal mass. If the aneurysm produces symptoms, it must be assumed that it is enlarging and in danger of rupturing. In these cases resection is indicated. Indeed, enlarging aneurysms, even in the absence of symptoms, progress toward rupture and should be removed.

The 3 new cases with chronic traumatic aneurysms of the thoracic aorta herein reported were asymptomatic. The diagnosis of traumatic aneurysm followed discovery on a chance roentgenogram of the chest of a mediastinal mass, which in 2 instances was calcified. The time that had elapsed between chest trauma and diagnosis varied from 7 years in Case II to 33 years in Case I. In contrast to these asymptomatic cases, Case IV, reported by Sarot and Lazzarini,[14] became symptomatic 14 years after trauma. Diagnosis in her case followed roentgenography of the thorax made because of severe cough and left shoulder pain. Garamella and associates[7] reported a fatal spontaneous rupture into the esophagus of a traumatic aortic aneurysm 8 years after trauma. Spencer *et al.*[16] also had a patient who was well for 14 years following an automobile accident and then showed enlargement of the aneurysm; surgical treatment was of no avail.

Because of enlargement of the aneurysm in Case III, resection was performed even though the patient was asymptomatic. Conservative management should be reserved for cooperative individuals whose chronic traumatic aneurysm has been present for many years. In these individuals routine periodic physical and roentgen examinations of the chest are essential in order to make certain that the aneurysm does not enlarge. Should symptoms of pain, hoarseness, cough, or hemoptysis develop, excision is advisable.[17] De Bakey,[3] however, recommends surgical treatment of every patient with a traumatic aneurysm, and this advice seems sound in the case of acute traumatic aneurysms even though very few patients with such aneurysms of the thoracic aorta have been saved by emergency operation.[12,15,16] However, it frequently offers the only chance of survival, and early diagnosis and surgical treatment are essential. The patient reported by Malm and Deterling[10] shows that even a case of an acute traumatic aneurysm of 4 months' duration which has progressed to heart failure, hypertension of the upper extremities, and decreased blood pressure in the legs (simulating coarctation of the aorta) can be salvaged by resection of the aneurysm and replacement by a graft.

Morris in the discussion of the paper by Spencer and associates[16] reported that the De Bakey group had resected 17 chronic traumatic aneurysms of the thoracic aorta without a fatality. With such reported results, resection and aorta repair should be considered even in cases of stabile long-standing chronic traumatic aneurysms.

SUMMARY AND CONCLUSIONS

In 3 asymptomatic male patients with left superior mediastinal masses, a chronic traumatic aneurysm of the thoracic aorta was diagnosed following a chance roentgenogram of the chest. Seven, nine, and thirty-three years, respectively, had elapsed since the occurrence of blunt thoracic trauma. In 2 instances the aneurysms developed after an automobile accident; in the third patient, after a fall from a 9 story window. In the latter case, resection of the aneurysm and replacement with a prosthetic graft using the atrial-femoral arterial bypass were done because serial roentgenograms of the chest demonstrated enlargement of the aneurysm although the patient was asymptomatic.

Operation was also advised in the other 2 cases since present-day left atrial-femoral

bypass techniques permit resection of aneurysms with a reasonably low mortality.[2,8] Due to the improved techniques, it is felt that salvage of patients with acute traumatic ruptures of the thoracic aorta is possible if the diagnosis is made early.

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ADDENDUM

Since this paper was submitted for publication, Dobell and colleagues have successfully repaired a traumatic rupture of the thoracic aorta (Dobell, A. R. C., MacNaughton, E. A., and Crutchlow, E. F. Successful early treatment of subadventitial rupture of the thoracic aorta. *New England J. Med.*, 1964, *270*, 410–412). Widening of the mediastinum and a left pleural effusion led to early diagnosis of rupture. With left atrial-femoral artery bypass, suture of the aortic tear, cross clamping of the aorta, resection of the rupture and replacement by a woven teflon prosthesis were achieved 39 hours after injury. These authors also cite Pessaro and Pace (*Surgery*, 1959, *46*, 787–791) who likewise repaired an aortic rupture on the day of injury; this case, however, had recurrence of a small fusiform aneurysm of the aortic arch. Fleischaker and coworkers have also recently successfully repaired acute traumatic ruptures of the aorta in 2 of 3 patients. (Fleischaker, R. J., Mazur, J. H. and Baisch, B. F. Surgical treatment of acute traumatic rupture of the thoracic aorta. *J. Thoracic & Cardiovascular Surg.*, 1964, *47*, 289–297.)

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ANEURYSM OF THE MEMBRANOUS SEPTUM*

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ANEURYSMS of the membranous septum are relatively rare lesions probably of congenital origin. Although in many cases they are of no clinical significance, these aneurysms may produce serious hemodynamic changes by obstructing the outflow tract of the right ventricle or as a result of rupture. The aneurysm may extend into and distort or replace the septal leaflet of the tricuspid valve. This can pose a potential complication at the time of open heart surgery if the approach to the right ventricle is through the tricuspid leaflet.

There is no clinical picture that is characteristic or even suggestive of a membranous septal aneurysm. In the absence of complications, the findings of cardiac catheterization, dye dilution studies, and so on, will be completely normal. Angiocardiography is the only technique by which a definite diagnosis can be made. As angiocardiography, particularly selective left ventriculography, is becoming more and more a part of the preoperative study of cardiac patients, it is important that the roentgenologic appearance of these aneurysms and their significance be appreciated.

ANATOMY AND PATHOLOGY

The membranous portion of the ventricular septum, although small in area, is of critical importance because of its intimate relationship to the aortic and atrioventricular valves, the two ventricles and the right atrium. There is some inconsistency in the use of the term "membranous septum" by different authors. In common usage, "membranous septum" often is meant to designate only the thin, fibrous portion of the *interventricular* septum. This excludes the posterior continuation of this thin septum which intervenes between the left ventricle and the right atrium. Such a division appears arbitrary. "Membranous septum" will be used in this paper in accord with the definition of Tandler[27] and others[2,10] to indicate the entire, thin portion of the septum which can be transilluminated.

The margin of the thick muscular septum as it faces the base of the heart is crescent-shaped and, on the left ventricular side, reaches the aortic valve only in its anterosuperior portion (Fig. 1 and 2). Posteriorly, the base of the muscular septum extends to the mitral annulus in the region of the posteromedial commissure. The portion of the ventricular septum between the concave rim of the muscular septum and the aortic valve is relatively thin and composed largely of fibrous tissue. This area comprises the membranous septum. It lies immediately beneath the anterior half of the noncoronary cusp and, depending on its size, extends a variable distance underneath the right coronary cusp.[8] Anteriorly, the membranous septum merges imperceptibly with the thick muscular septum. Posteriorly, the septum is continuous with the dense right fibrous trigone and the annulus fibrosus of the mitral valve.

Both the tricuspid and mitral valves attach, in part, to the ventricular septum but at different levels. The attachment of the septal leaflet of the tricuspid valve is anterior to the anterior leaflet of the mitral valve (Fig. 1). The line of attachment of the septal leaflet of the tricuspid valve crosses the membranous septum while the attachment of the anterior leaflet of the mitral

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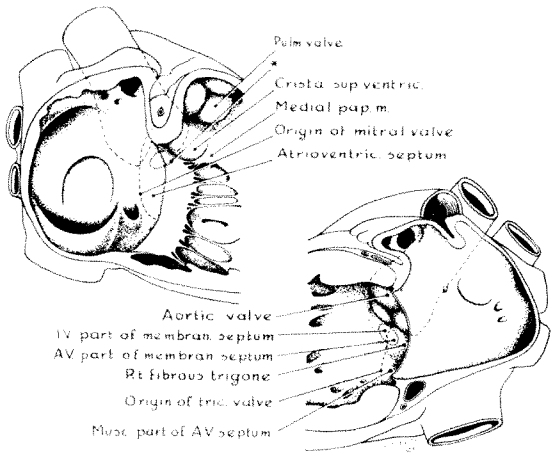


FIG. 1. Drawings illustrating the relationship of the base of the ventricular septum to the mitral, aortic and tricuspid valves. The upper figure shows the appearance of the right side of the septum, the lower figure the left. The dotted ellipse represents the thinnest portion of the membranous septum. On the right side, a portion of the membranous septum (*) is hidden by the tricuspid valve. Note that the tricuspid attachment crosses the membranous septum while the attachment of the mitral valve lies behind it.

valve is entirely posterior to the membranous septum (Fig. 2). Therefore, the portion of the membranous septum anterior to the tricuspid valve lies between the ventricles while the portion of the septum posterior to the tricuspid valve is between the left ventricle and the right atrium, forming a part of the so-called atrioventricular septum.

An aneurysm of the membranous septum may project into the right ventricle, into the septal leaflet of the tricuspid valve, or into the right atrium. Rarely, the aneurysmal sac may extend upward and appear on the exterior of the heart adjacent to the aorta.[12,14] The mouth of the aneurysm is localized and sharply defined and, in the gross specimen, has an appearance identical to a "membranous ventricular septal defect." The opening into the aneurysm most commonly lies immediately beneath the commissure between the right coronary and noncoronary aortic cusps (Fig. 3, *A*, *B* and *C*; and 4, *A* and *B*). These two cusps and the commissure then form a tent-like roof of the aneurysm. In some cases, the aneurysm arises completely in front of or behind this commissure. The appearance of the aneurysm on the right side of the septum will depend largely on the size of the sac and the course of the aneurysm. In general, the aneurysm is no larger than 1 to 2 cm. in all diameters although one measuring 6 cm. in length and with a capacity of 12 cc. has been recorded in the literature.[12] The

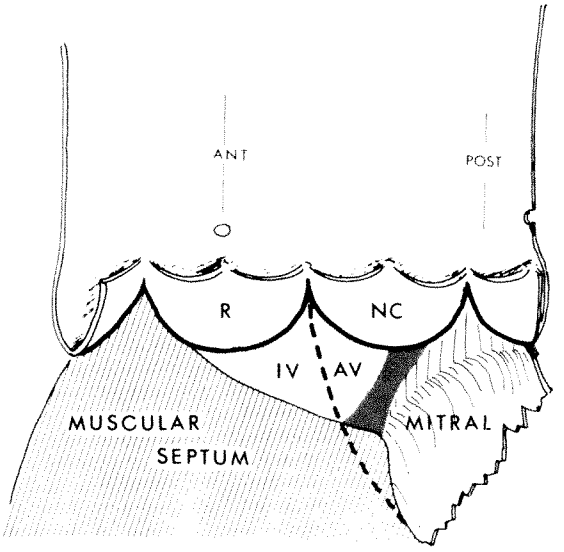


FIG. 2. A diagrammatic sketch of the basal portion of the septum as seen from the left ventricular side. The upper margin of the muscular septum is crescent-shaped and extends from below the right coronary cusp (R) posteriorly to the attachment of the mitral valve. The clear area represents the membranous septum which is divided by the attachment of the septal leaflet of the tricuspid valve (dashed line) into an interventricular (IV) and an atrioventricular (AV) portion. Anteriorly, the membranous septum merges imperceptibly (lightly stippled area) with the muscular septum and the root of the aorta. Posteriorly, the membranous septum is continuous with the right fibrous trigone (heavily stippled area). The anterior leaflet of the mitral valve is attached to the root of the aorta along the posterior portion of the left coronary cusp and the adjacent portion of the noncoronary cusp (NC). The attachment continues along the right fibrous trigone and the posterior margin of the base of the muscular septum. Note that the atrioventricular septum is formed in part by the membranous septum and in part by the muscular septum. The anterior (Ant) and posterior (Post) margins of the aorta *in situ* are indicated.

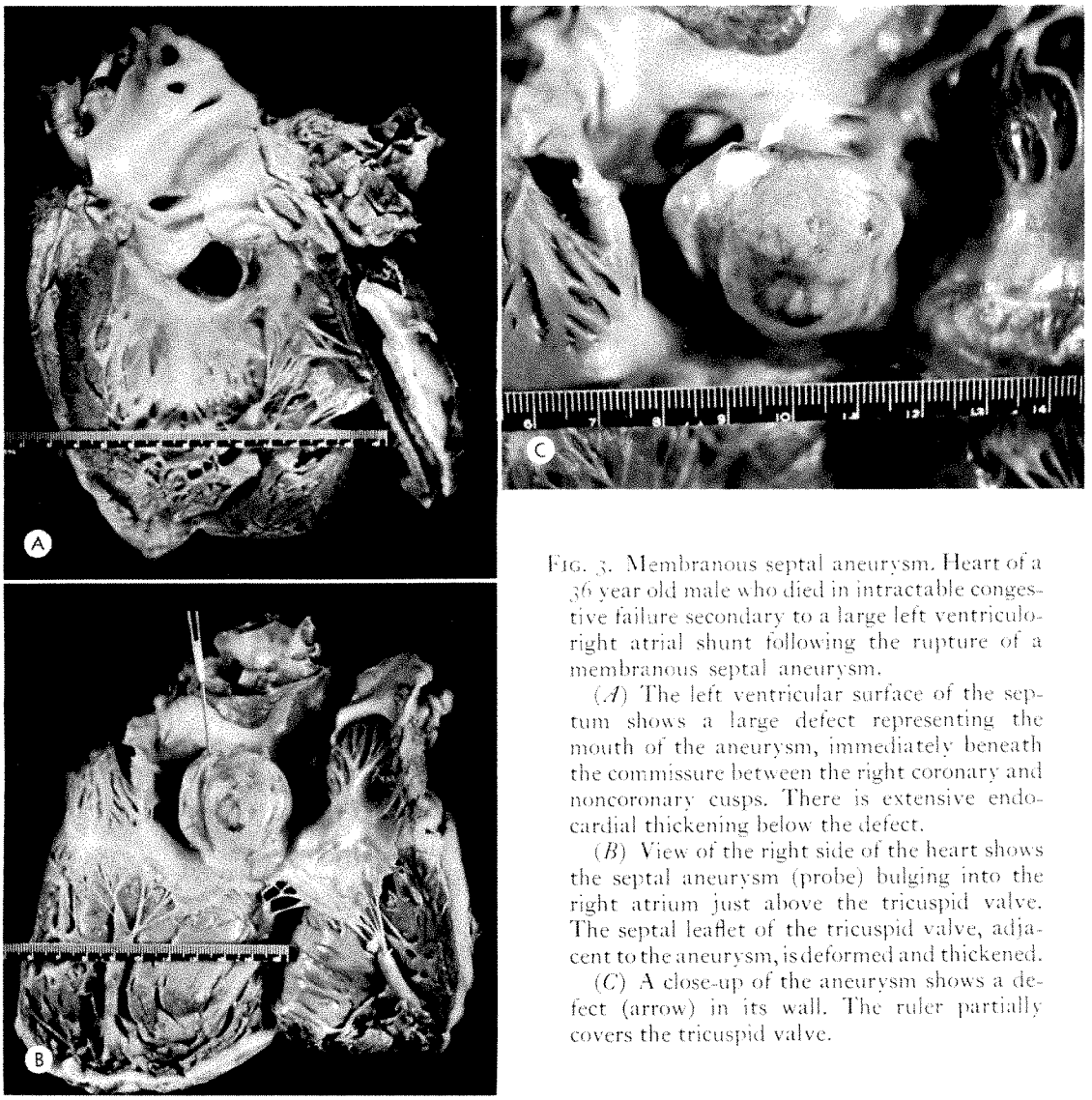


FIG. 3. Membranous septal aneurysm. Heart of a 36 year old male who died in intractable congestive failure secondary to a large left ventriculo-right atrial shunt following the rupture of a membranous septal aneurysm.

(*A*) The left ventricular surface of the septum shows a large defect representing the mouth of the aneurysm, immediately beneath the commissure between the right coronary and noncoronary cusps. There is extensive endocardial thickening below the defect.

(*B*) View of the right side of the heart shows the septal aneurysm (probe) bulging into the right atrium just above the tricuspid valve. The septal leaflet of the tricuspid valve, adjacent to the aneurysm, is deformed and thickened.

(*C*) A close-up of the aneurysm shows a defect (arrow) in its wall. The ruler partially covers the tricuspid valve.

most common site of presentation on the right side of the heart is in the right ventricle[5,7,11] below and behind the crista supraventricularis immediately adjacent to the septal leaflet of the tricuspid valve. The aneurysm may elevate this leaflet[5] and protrude into the tricuspid orifice[6,11] or it may extend superiorly and anteriorly toward the outflow tract of the right ventricle[19] (Fig. 4, *A* and *B*). In some cases, the aneurysm may extend directly into the base of the tricuspid valve and replace a portion of the anterior or septal leaflet.[13,22] When the aneurysm is formed from the atrioventicular septum, it may present in the right atrium above the tricuspid valve (Fig. 3, *A*, *B* and *C*).[5,15,20]

The wall of the aneurysm is composed of fibrous tissue. The interior of the sac, *i.e.*, the left ventricular side, is usually lined with thickened fibrous trabeculae resembling chordae tendineae (Fig. 4, *A* and *B*). Some of these appear to limit the distensibility of the sac which bulges around them and gives the aneurysm a multilocular appearance.

Aneurysms of the membranous septum often occur as isolated, uncomplicated de-

fects which are found incidentally at the time of autopsy. Not uncommonly, however, the aneurysm has a defect in its wall producing a left to right shunt.[18,19,24] In some of these cases, the murmur due to the ventricular septal defect was first detected in early childhood, suggesting that the defect was of congenital origin.[24] In other cases, the defect possibly represents a small rupture of an enlarging aneurysm. We have seen one patient (Fig. 3, *A*, *B* and *C*) in whom rupture of a membranous septal aneurysm produced a sudden, large, left ventriculo-right atrial shunt and caused the demise of the patient. Examination of the heart at autopsy showed bacterial endocarditis of the wall of the aneurysm, presumably the cause of the rupture.

The most common cardiac lesion associated with a membranous septal aneurysm is aortic valvular insufficiency. This may be due to prolapse of an aortic cusp into the mouth of the aneurysm.[7] The mechanism is similar to that which occurs with membranous ventricular septal defects.[25] Occasionally, the right coronary or noncoronary cusp is thick and shortened[20,22] and may show multiple fenestrations.[5,12] Membranous subvalvular aortic stenosis,[20] defects of the atrial septum,[13,21,31] complete transposition,[16] and various degrees of heart block[6,7] have also been reported in association with aneurysms of the membranous septum. An aneurysm may be the site of bacterial endocarditis[14,21] or thrombus formation.[9]

One group of aneurysms in the region of the membranous septum can be distinctly separated from the others; namely, those associated with the various forms of persistent common atrioventricular canal.[18] In these cases, the membranous and atrioventricular septa do not develop normally and are usually completely absent together



FIG. 4. Membranous septal aneurysm producing right ventricular obstruction. Clinically, the patient was considered to have pulmonic stenosis.

(*A*) The left ventricular side of the septum shows the mouth of the aneurysm to involve almost the entire membranous septum. The tent-like roof of the aneurysm is formed by the commissure between the right coronary and noncoronary cusps. Multiple, abnormal chordae can be seen within the lower part of the aneurysm.

(*B*) View from above. The outflow tract of the right ventricle extending from the crista supraventricularis (CS) to the pulmonic valve (PV) has been opened. The aneurysm (arrow) can be seen in the right ventricle immediately below the crista, bulging into and partially obstructing the mouth of the infundibulum.

with the crest of the muscular septum.[28] The anterior leaflet of the mitral valve is cleft and its line of attachment to the ventricular septum is greatly lengthened as contrasted to the normal. The superior segment of the cleft mitral leaflet is attached to the septum by abnormal chordae tendineae which fill the area normally occupied by the membranous septum.[30] The chordae are connected by thin fibrous tissue which may bulge between them and form an aneurysm extending into the right ventricle or into the common atrioventricular orifice. The abnormal attachment of the anterior mitral leaflet extends along the deficient, "scooped-out" crest of the muscular septum so that an aneurysm in this instance may form at some distance from the aortic valve.

ROENTGEN FINDINGS

Uncomplicated aneurysms of the membranous septum do not cause any change in the size or conformation of the cardiac silhouette. Abnormalities of the heart contour, when they are present, are due to hemodynamic alterations produced by associated lesions or by complications due to aneurysm itself. The levocardiogram phase of a venous or selective right sided angiocardiogram may demonstrate the aneurysm[10,26] if it is large. In the majority of cases, the greater anatomic detail available with left ventricular angiocardiography will be necessary for clear delineation.

As the various parts of the basal portion of the ventricular septum cannot be distinguished on an angiocardiogram, it is necessary to localize these areas in terms of their relationship to the aortic cusps.[3] In the frontal projection of a left ventricular angiocardiogram, the right coronary cusp is seen *en face*. The noncoronary cusp is viewed tangentially along the right border of the aortic valve. The right border of the ventricular outflow tract from the aortic valve to the posteromedial mitral commissure is normally exquisitely smooth and straight. The membranous septum forms the upper part of this contour. In the lateral view, the right coronary cusp is projected tangentially along the anterior border of the aorta while the noncoronary cusp is seen essentially *en face* as the most inferior of the aortic cusps. The anterior margin of the left ventricular outflow tract, in this view, is formed mainly by the conus portion of the ventricular septum. An unusually large membranous system may, however, contribute to the uppermost portion of this contour.

An aneurysm of the membranous septum communicates with the left ventricle and is opacified with the left ventricle. On the left ventriculogram, it is seen as a saccular, sometimes lobulated, outpouching of the left ventricular cavity immediately below the aortic valve (Fig. 5, *A* and *B*; 6, *A* and *B*; 7, *A* and *B*). The projection in which the aneurysm is best visualized will depend on the location of its mouth and the course of the aneurysm. When the aneurysm is beneath the right coronary cusp, it will usually extend anteriorly and be seen best in the lateral projection (Fig. 5*A*), protruding into the right ventricle immediately below the crista supraventricularis (Fig. 6*A*). Since the right coronary cusp is seen *en face* in the frontal view, the aneurysm may be completely obscured in this projection. An aneurysm arising beneath the noncoronary cusp will extend downward and to the right from beneath the right margin of the aortic valve and will be more evident on the frontal than the lateral projection (Fig. 7, *A* and *B*).

If there is a defect in the aneurysm, a shunt from the left ventricle to the right ventricle (Fig. 5, *A* and *B*; 6, *A* and *B*; and 7, *A* and *B*) or to the right atrium (Fig. 8, *A* and *B*) will be demonstrated on the left ventricular angiocardiogram. If the defect in the aneurysm is sufficiently large in relation to the size of the aneurysm, the saccular appearance may be lost. We have seen 2 cases of ventricular septal defects, studied by left ventriculography, in which the stream of contrast material crossing the membranous septum first narrowed before it dispersed in the right ventricle. It is pos-

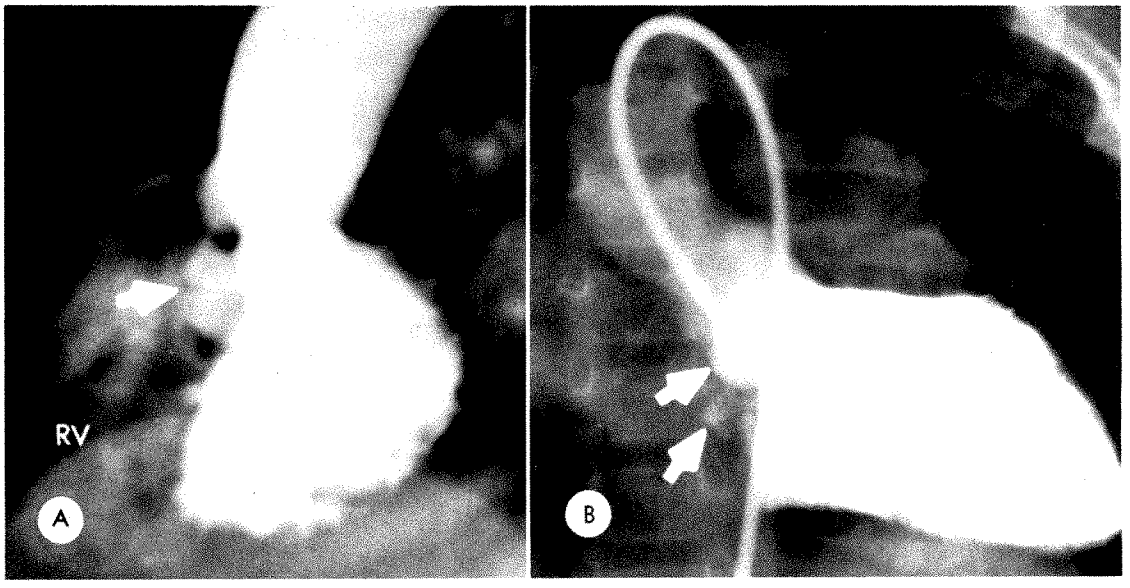


FIG. 5. Membranous septal aneurysm, left ventricular angiocardiograms.

(*A*) Lateral projection. The membranous septal aneurysm is seen as a localized, lobulated bulge (arrow) extending forward from the left ventricle beneath the right coronary cusp. The right ventricle (RV) is faintly opacified indicating the presence of a left to right ventricular shunt.

(*B*) Frontal projection. An early roentgenogram shows the left ventricle filled with contrast material. The aorta is faintly opacified. The aneurysm can be identified as a localized collection of opaque material (upper arrow) along the right border of the outflow tract. A jet (lower arrow) arises from the lower part of aneurysm. Note that the aneurysm simulates a normal sinus of Valsalva but is located adjacent to the muscular septum at a lower level than the aortic valve.

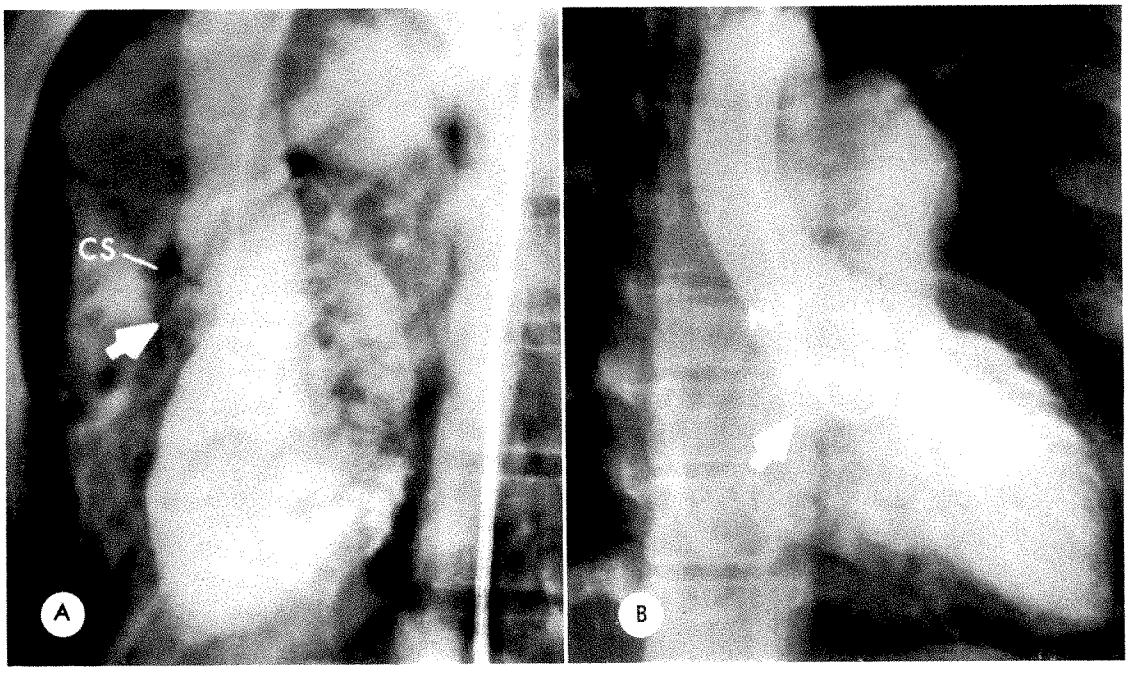


FIG. 6. Membranous septal aneurysm, left ventricular angiocardiograms.

(*A*) Lateral projection. The aneurysm (arrow) bulges into the right ventricle below the crista supraventricularis (CS). A left to right interventricular shunt is present.

(*B*) Frontal projection. The aneurysm (arrow) in this view presents as a slight, localized bulging of the left ventricular contour immediately below the noncoronary cusp (N).

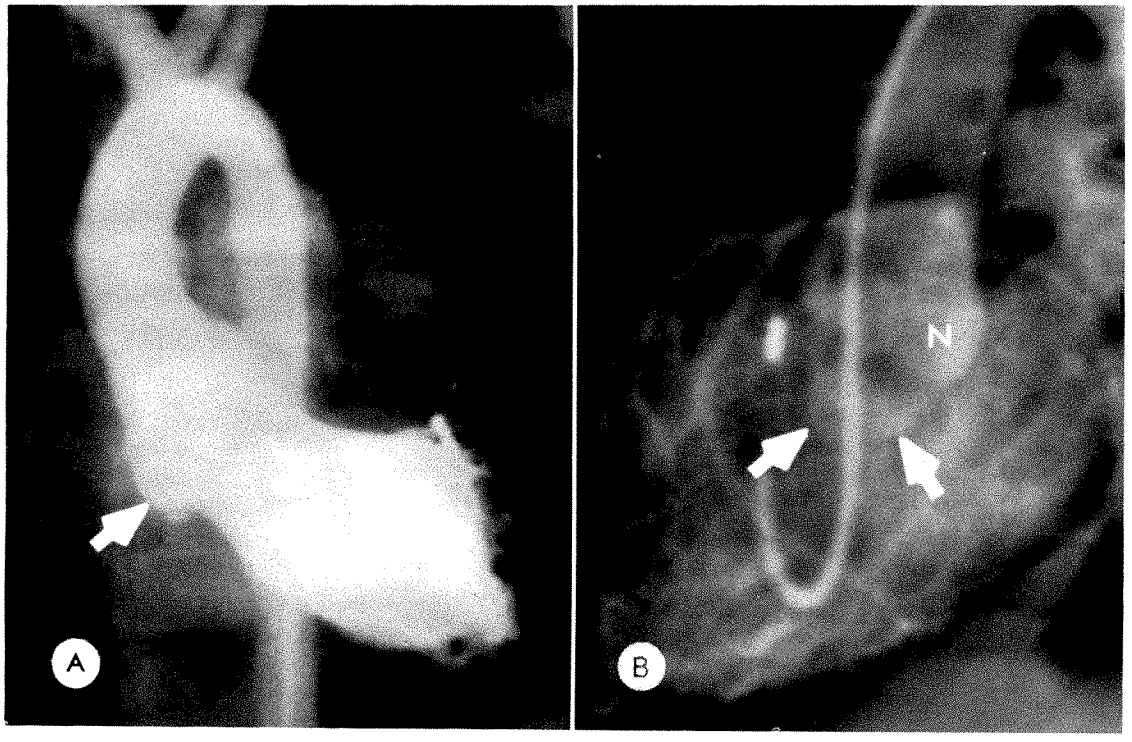


Fig. 7. Membranous septal aneurysm, left ventricular angiocardiograms.

(*A*) Frontal view. The membranous septal aneurysm (arrow) presents as a localized bulge along the upper right contour of the left ventricle.

(*B*) Lateral projection. A late roentgenogram with almost complete emptying of the left ventricle shows a residual collection of contrast material in the aneurysmal sac (arrows). This aneurysm is located posteriorly beneath the noncoronary cusp (N) and did not project beyond the anterior border of the left ventricle. As a result, when the ventricle was filled with opaque material the aneurysm was obscured.

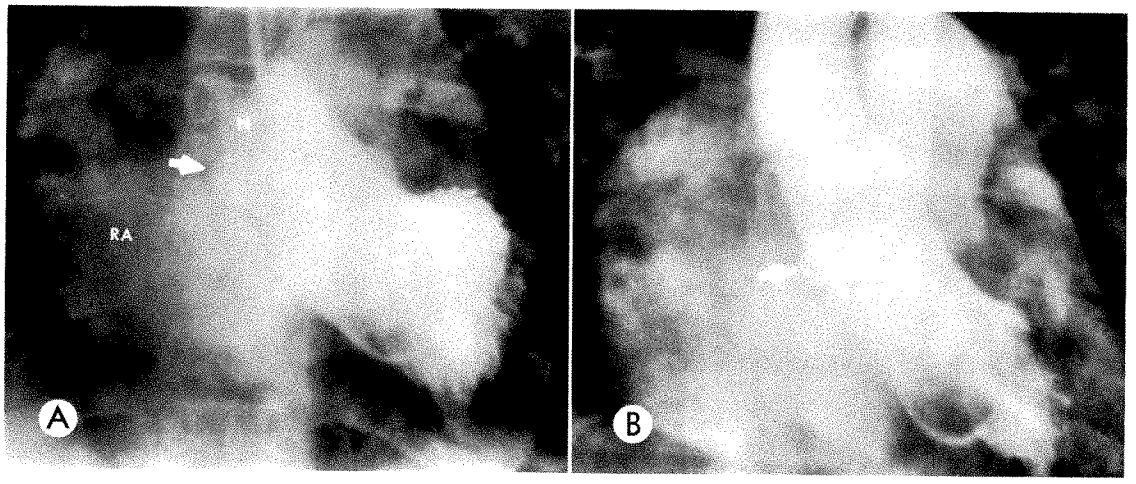


Fig. 8. Membranous septal aneurysm, left ventricular angiocardiograms.

(*A*) Frontal projection. Early roentgenogram before filling of the aorta. There is an aneurysmal bulge (arrow) of the left ventricle below the noncoronary cusp (N). The right atrium (RA) is opacified as a result of a left ventriculo-right atrial shunt.

(*B*) A later roentgenogram from the same series shows the aneurysm (arrow) to better advantage. The right ventricle and pulmonary artery are now opacified.

sible that this narrowing of the stream was produced by the residual walls of a septal aneurysm with a relatively large defect at its apex. Anatomic proof in these cases is not available.

In the presence of an endocardial cushion defect, the anatomy and function of the cleft anterior leaflet of the mitral valve are well demonstrated on the frontal projection of the left ventricular angiocardiogram. A detailed description of these findings has been published elsewhere.[4] During systole, the superior and inferior segments of the cleft mitral leaflet bulge towards the left atrium and produce a scalloped contour to the right border of the left ventricular outflow tract. In diastole, as nonopaque blood from the left atrium enters the ventricle, the valve leaflets fold into the ventricle. The scooped-out crest of the muscular septum is then visible and the left ventricular outflow tract appears elongated and narrowed. In the lateral projection, instead of the normally smooth anterior left ventricular contour, the attachment of the abnormal anterior mitral leaflet to the septum beneath the right coronary and noncoronary cusps produces scalloping of this margin. Aneurysms can occur anywhere along the attachment of the anterior mitral leaflet to the septum. In general, they are best visualized in the lateral projection, extending anteriorly (Fig. 9, *A* and *B*). The aneurysm may interfere somewhat with the mobility of the cleft mitral segments but not to a degree great enough to obscure the roentgen characteristics of the endocardial cushion defect.

The lesion most likely to be confused with a membranous septal aneurysm on the left ventricular angiocardiogram is an aneurysm of a sinus of Valsalva. Sinus aneurysms often extend through associated ventricular septal defects and present on the right side of the heart in the same locations as membranous septal aneurysms.[23] Left ventricular angiocardiography may not be sufficient to differentiate between these two lesions unless the exposures are so fortuitous as to show complete filling of the

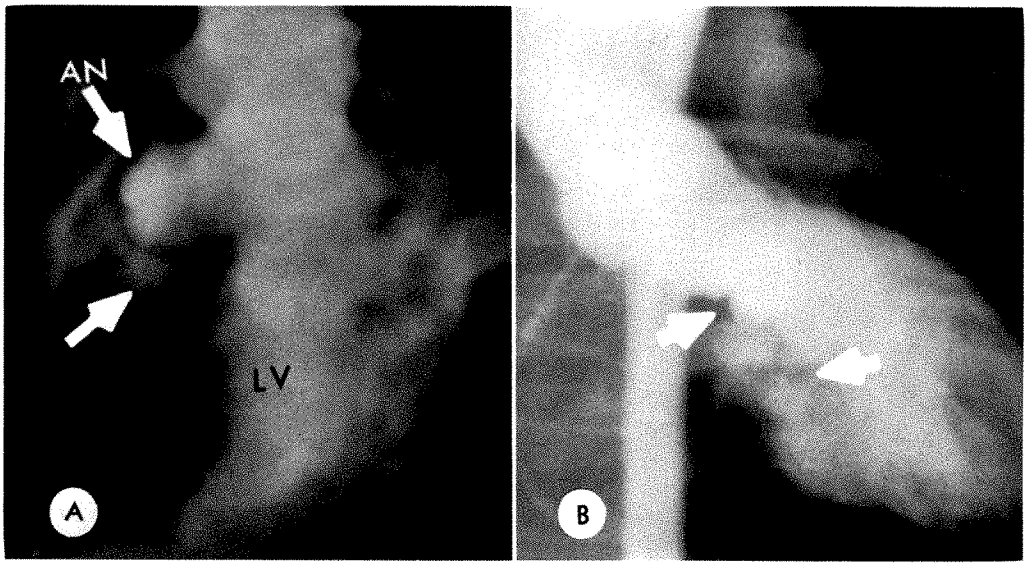


FIG. 9. Septal aneurysm associated with an endocardial cushion defect, left ventricular angiocardiograms.

(*A*) Lateral projection. A large aneurysm (AN) projects anteriorly beneath the right coronary cusp. Contrast material in the right ventricle (arrow) outlines the thickness of the wall of the aneurysm. (Reproduced with permission from *Radiology*.)

(*B*) Frontal projection. The aneurysm cannot be identified in this view. Scalloping of the upper right contour of the outflow tract (upper arrow) is produced by the abnormal attachments of the superior segment of the cleft anterior mitral leaflet. The coapted margins of the two segments of this leaflet produce a faint, linear lucency (lower arrow).

left ventricle before any contrast material enters the aorta (Fig. 5, *A* and *B*; and 8, *A* and *B*). If the aneurysm fills with the left ventricle, it must arise in the infravalvular region whereas a sinus of Valsalva aneurysm will fill only after the aorta is opacified. A second injection in the supravalvular region of the aorta will be conclusive unless marked aortic insufficiency is present.

In several instances, left ventricular angiocardiography has shown localized projections of the left ventricle (Fig. 10) in the region of the membranous septum which are smaller and more irregular than the aneurysms described above. In one such case operated upon for relief of infundibular stenosis, an aneurysm could not be identified through the right ventriculotomy. Palpation of the atrial septum and the tricuspid valve also failed to reveal any abnormality. The nature of these pockets is not entirely clear, but it is possible that they represent an early stage in the development of an aneurysm.

The appearance of a membranous septal aneurysm can be mimicked by a simple ventricular septal defect when the septal leaflet of the tricuspid valve is abnormally adherent to the right side of the defect.[17] Contrast material can pass through the septal defect and be pocketed by the deformed tricuspid valve. In the lateral view, this pocket will appear as a projection from the septum at a distance below the aortic valve.

An aneurysm of the muscular septum may develop following a septal myocardial infarction.[1,29] This aneurysm will bulge into the right ventricle but can be differentiated from a membranous septal aneurysm by its location closer to the apex. In the one case we have studied by angiocardiography, the aneurysm was seen only in the frontal projection, bulging from the right border of the left ventricle below the posteromedial commissure of the mitral valve (Fig. 11).

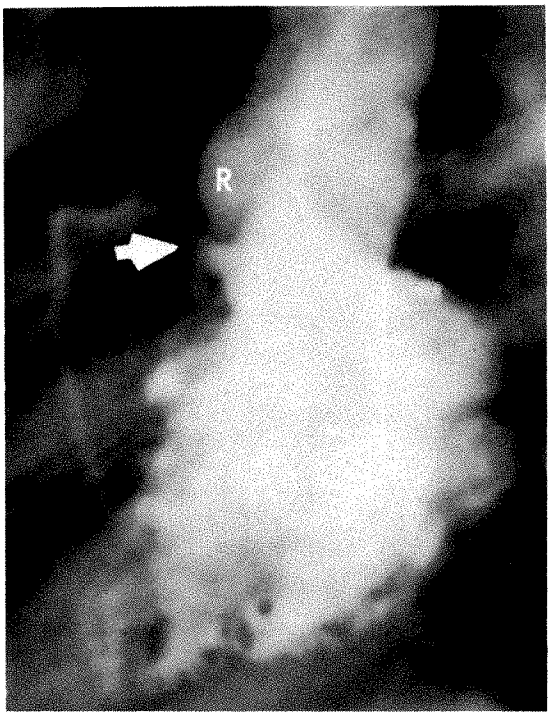


Fig. 10. Left ventricular angiocardiogram. Lateral view. A small, angular projection (arrow) of the left ventricle extends anteriorly below the right coronary cusp (R). The nature of this projection is uncertain since it was not detected during the right ventricular surgery for infundibular stenosis.

DISCUSSION

Aneurysms of the membranous septum are most likely congenital in origin. However, these lesions have not been described, to our knowledge, in fetal or newborn hearts. This suggests that the aneurysms develop some time after birth in response to the normal, high left ventricular pressure although the basic weakness in the septum is embryologically determined. The exact defect in cardiac development that produces this weakness is not certain. The most widely accepted theory to date is that proposed by Mall.[15] He noted, in reviewing several cases, that the aorta seemed to arise astride the muscular septum, rather than entirely from the left ventricle. As a result, the membranous septum was oriented in an almost horizontal plane rather than in its normal vertical position. Mall felt that this shift in position caused a weakening of the membranous septum which made it susceptible to aneurysmal dilatation. We have not been able to demonstrate an abnormal relationship between the aorta and the ventricular septum by

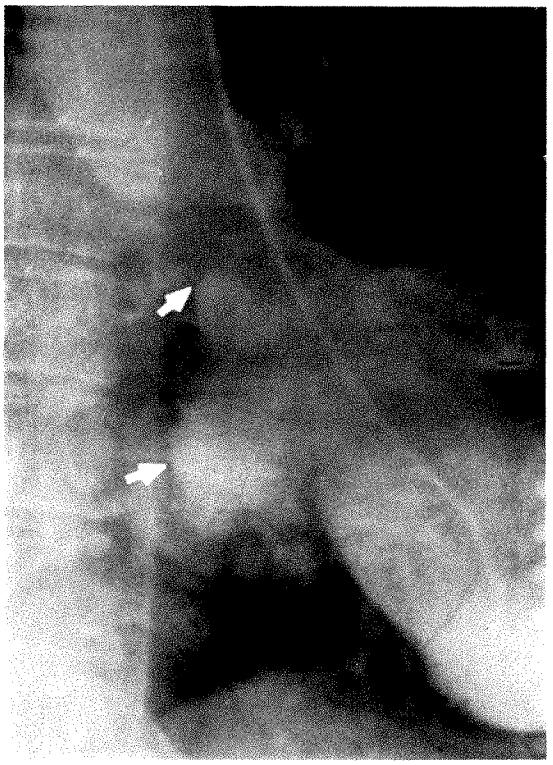


FIG. 11. Aneurysm of the muscular septum following a myocardial infarction. Left ventricular angiocardiogram. Frontal view. A localized bulge (lower arrow) of contrast material projects into the right ventricle from the region of the posteromedial mitral commissure. The aneurysm is at some distance from the aortic valve (upper arrow). A small, left to right interventricular shunt is present.

angiocardiography or in 2 autopsied cases. Other authors have speculated that these aneurysms originate as a result of defective fusion of the endocardial cushions[11,22] or of the conus ridges with the endocardial cushions and the muscular septum.[23] It should be noted that the membranous septum, when first formed in the embryonic heart, is composed of bulky myocardium and not fibrous tissue. It is only at a later stage in development that this portion of the septum becomes fibrous. An abnormality in the process in which the myocardial tissue disappears may result in an unusually thin or unusually large and weak membranous septum. This would then represent the developmental basis for the formation of an aneurysm.

An aneurysm of the membranous septum always bulges towards the right side of the heart presumably because of the higher pressure in the left ventricle. We have seen such a lesion produced artificially in a patient following repair of a membranous septal defect. Because of severe pulmonary hypertension, a perforated patch of compressed Ivalon was used to bridge the defect. Restudy several years after surgery showed the patch in the region of the membranous septum, bulging into the right ventricle and producing an appearance identical to that of a septal aneurysm (Fig. 12). Catheterization at this time showed the right ventricular pressure to be considerably less than systemic pressure. The role of differential ventricular pressure in the formation of a membranous septal aneurysm is further indicated by the appearance of an aneurysm in a heart, in our collection, with corrected transposition of the great vessels. The mouth of the aneu-

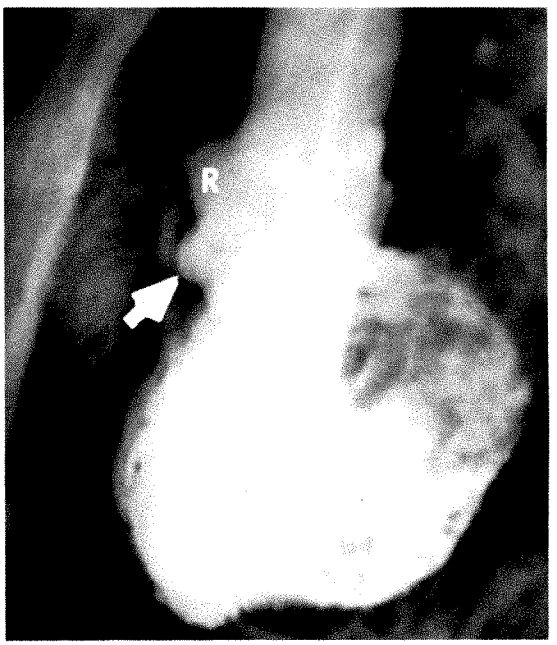


FIG. 12. Iatrogenic membranous septal aneurysm. Left ventricular angiogram made several years after repair of a membranous septal defect with a perforated patch. Lateral projection. The patch (arrow) bulges anteriorly beneath the right coronary cusp (R) and simulates an aneurysm of the membranous septum. A small, residual interventricular shunt is present.

rysm was inferior to the crista supraventricularis in the morphologic right (or systemic) ventricle, the usual location of the membranous septum. However, in this case, the aneurysm bulged into the morphologic left ventricle (from which the pulmonary artery arose). Cardiac catheterization showed the pressure in the morphologic right ventricle to be higher than the left.

An aneurysm of the membranous septum is a surgically correctable lesion.[19,21] When approached from the right side of the heart, the aneurysm can be excised at its base and the defect in the septum closed. This procedure is to be recommended when the aneurysm is producing significant hemodynamic changes. If growth of the aneurysm can be demonstrated on serial angiocardiograms, surgical correction may also be indicated.

SUMMARY

1. Aneurysms of the membranous septum are relatively rare lesions presumably developing on a congenital basis. These aneurysms originate in the left ventricle immediately beneath the aortic valve and bulge into the right ventricle, the septal leaflet of the tricuspid valve or into the right atrium.

2. Many of these aneurysms do not produce symptoms. Others may cause right ventricular outflow tract obstruction or may rupture and result in a septal defect. The resulting shunt will be from the left ventricle into the right atrium or ventricle.

3. An aneurysm of the membranous septum may be an isolated abnormality or be associated with other congenital cardiac defects, particularly aortic valvular insufficiency. Membranous septal aneurysms may be the site of bacterial endocarditis or thrombus formation.

4. Aneurysms resembling those arising from the membranous septum may occur as part of the complex of deformities produced by an endocardial cushion defect. However, the angiographic features diagnostic of a cushion defect can still be recognized.

5. The presence of a septal aneurysm can be established only by angiocardiography. The diagnostic features as seen on the left ventricular angiocardiogram are presented.

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VOL. 91, NO. 6

ANEURYSMS OF THE CORONARY ARTERY

A CASE REPORT

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ANEURYSM of the coronary vessels of the heart was first reported by Bougan in 1812. Since that time, and prior to a recent and comprehensive study by Daoud, Pankin, Tulgan and Florentin,[1] this entity has been believed to be relatively uncommon.

In an excellent review and tabulation of the world medical literature, Daoud *et al.*[1] considered all previously reported cases of coronary artery aneurysm and added 10 new cases of their own. The only other detailed review on the subject was made in 1929 by Packard and Wechsler[3] who added but a single new case. The 10 new cases reported by Daoud *et al.* resulted from a detailed and careful examination of the hearts of 694 cases at autopsy at the Albany Medical College during the period from March 1955 to September 1957.

It is not within the scope of this brief communication to deal with the controversial pathogenesis of this entity. The interested reader is referred to a paper by Holman and Peniston,[2] who, at some length, discuss the results of clinical and experimental findings in relation to pathogenesis.

Daoud *et al.* in the conclusion of their paper state that aneurysms of the coronary vessels are probably not so rare as had been previously supposed; by the addition of 10 new cases, they increased the grand total of all reported cases by 12.6 per cent. They also state that in all of their cases the aneurysms were of arteriosclerotic origin, the microscopic examination in most instances revealing focal calcification.

Although the presence of calcification in the walls of coronary vessels is not unusual, our search of the radiologic literature failed to reveal any case in which a diagnosis of aneurysm of the coronary vessels was made on the basis of calcification in the wall of the aneurysm found on conventional roentgenograms of the chest.

REPORT OF A CASE

N.D., a 54 year old white male laborer, was admitted to the medical service of the Pottsville Hospital in Pottsville, Pennsylvania, for the first time on July 24, 1961 because of substernal chest pain, severe shortness of breath and extreme weakness. Since 1956 he had exertional and occasional nocturnal dyspnea. Three months before admission he had a sudden substernal pain with dyspnea and pain in both arms of 1 hour's duration, requiring no medical care. The next day he felt better and went to work as usual. Six weeks following this episode he had a sudden attack of aphasia with paresis involving the left arm and leg without unconsciousness, regaining speech after a few hours. After 1 month the paresis disappeared; however, he developed increasingly severe exertional chest pain and dyspnea which forced him to stop work. On the day of admission nocturnal dyspnea, extreme weakness and profuse perspiration occurred with a brief period of unconsciousness and he was referred for hospital care. He had had no serious childhood disease. The tonsils and adenoids were removed at 6 years of age with good result. Pneumonia occurred at 16 years of age, with no complication. Other general systems were unremarkable. For many years he had been an alcoholic (spree drinking type). He was a heavy cigarette smoker (2 or 3 packs daily).

Family History. Father died of coronary heart disease at 41 years of age; mother at 62 years of age of same disease. An elder brother had a myocardial infarction at 46 years of age. Three younger brothers and sisters are alive and well.

Physical Examination. This revealed a seriously ill male, appearing older than his age. He was orthopneic, cyanotic, and extremely apprehensive. The temperature was 97° F., axillary; pulse 154/min., regular rhythm; respira-

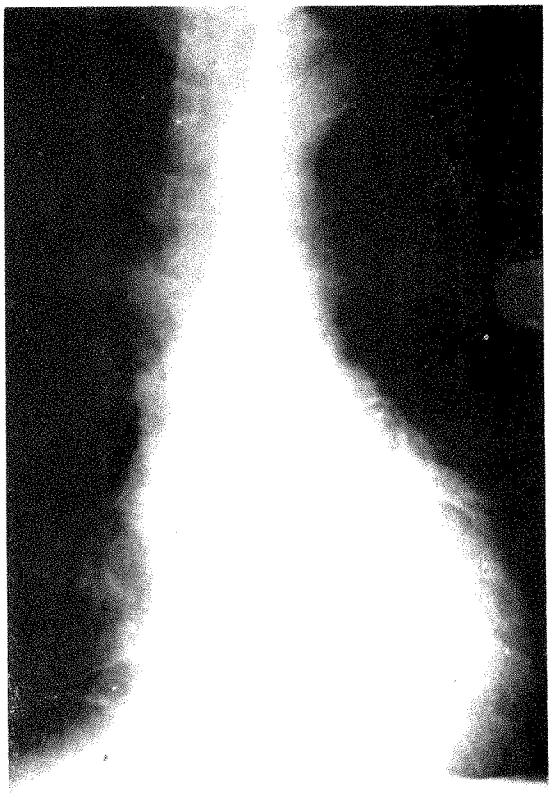


FIG. 1. Posteroanterior roentgenogram of chest shows ring shadows in right cardiophrenic angle (the largest measuring 3.2 cm. in diameter), representing calcification in the walls of multiple aneurysms of the right coronary vessel. Smaller ring shadows in left hilar area show additional involvement of the left coronary artery.

tions 34/min.; blood pressure 230/130. He was semiconscious and his skin was excessively moist and cold. There was no peripheral edema, the fingertips were slightly clubbed and coarse tremors were present. The skull, scalp, ears, nose, mouth, and throat were not significantly abnormal. The pupils were constricted and equal; the reflexes were sluggish. Arcus senilis and a Group 2 hypertensive retinopathy were present; the neck veins were distended and the carotid pulsations were low in volume with no bruit. The thorax was normal in shape, and there was impaired resonance over both lung bases with diffuse coarse moist rales. There were no palpable heart thrills; the apex beat was felt just outside the left mid-clavicular line in the fifth interspace. The first apical sound was faint, the second sound accentuated. A Grade 3 high pitched holosystolic apical murmur was present. The aortic component of the second sound was accentuated at the base; no basal murmurs were heard and there was no friction rub. The liver border was smooth and firm at the right costal margin. The abdomen was moderately distended with no masses or dullness in the flanks. The extremities presented no deformity and no venous varicosities. The peripheral vessels were thickened. The pulses were of equal force in both wrists and feet. The neurologic examination was not significantly abnormal.

Laboratory Findings. At admission, hematocrit was 51 per cent, sedimentation index 34 mm./hr., hemoglobin 17.6 gm., red blood cell count 5,610,000, white blood cell count 20,700 with 91 per cent neutrophils, and 9 per cent lymphocytes. Prothrombin time was 16 seconds, fasting blood sugar 96 mg. per cent, urea nitrogen 14 mg. per cent. Transaminase SGOT was 175 units; on July 25, 1961, it was 85 units. Urinalysis was negative; VDRL was negative. Transaminase was 80 units on July 26, 1961 and 25 units on July 27, 1961. On September 2, 1961, hemoglobin was 13.8 gm., red blood cell count 4,620,000, white blood cell

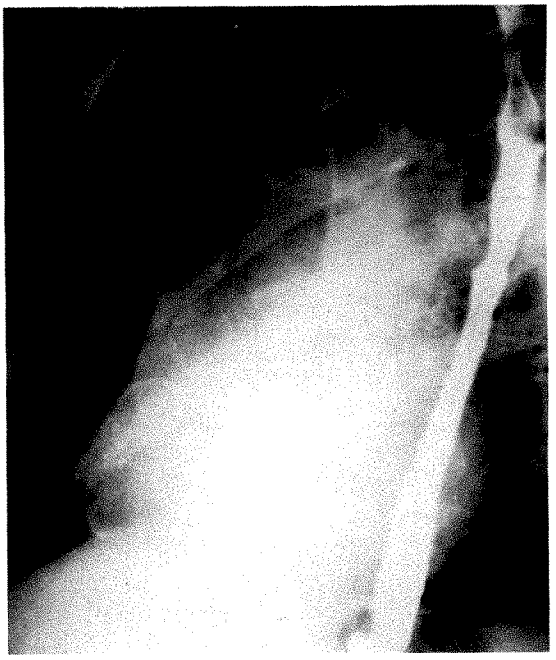


FIG. 2. Oblique study of cardiac silhouette shows the huge aneurysms of the right coronary vessel to better advantage.

count 7,400, with 63 per cent neutrophils, 32 per cent lymphocytes, 3 per cent monocytes, and 2 per cent eosinophils. The hematocrit was 42 per cent and the sedimentation index 38 mm./hr.

Electrocardiographic Findings. The electrocardiogram on admission revealed a supraventricular tachycardia and ventricular rate of 142 per minute with left ventricular hypertrophy. Subsequent electrocardiograms on July 25, 26 and 27 showed abnormalities compatible with an acute anterobasal myocardial infarction, a first degree atrioventricular heart block and digitalis effect. On August 17, 1961 the electrocardiogram revealed normal voltage T waves in V1, 2, 3 and 4, a return to regular sinus rhythm, a ventricular rate of 80 per minute and left ventricular hypertrophy with digitalis effect.

Clinical Course. The patient's temperature of 99.3° F. persisted for 4 days following admission. He was immediately digitalized with cedilanid followed by digoxin orally. He was heparinized by lipo hepin subcutaneously. Oral anticoagulants were withheld because of a prothrombin time of 64 seconds on August 12, 1961 with 0 per cent prothrombin activity. Digoxin was discontinued on August 13, 1961.

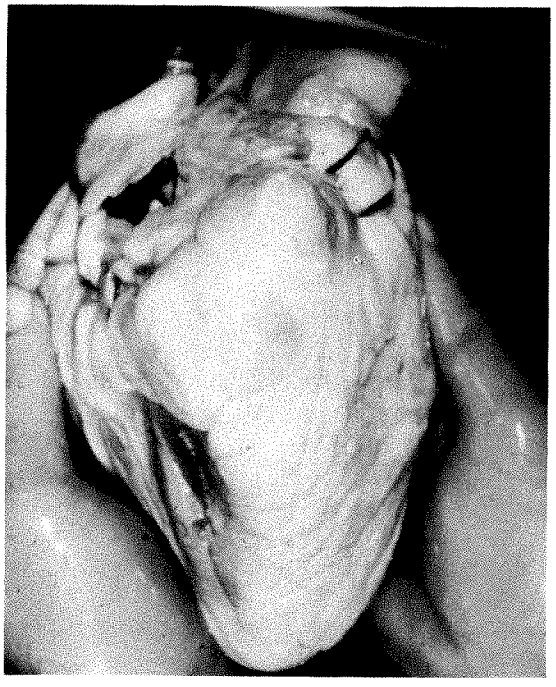


Fig. 3. Heart specimen shows nodulations along course of right coronary artery.



Fig. 4. Right coronary artery showing multiple aneurysms.

Chest roentgenograms (Fig. 1 and 2) were made on August 24, 1961 and the patient was discharged on September 2, 1961 to convalesce at home.

He had 3 subsequent admissions on December 27, 1962, February 18, 1963 and May 4, 1963 for treatment of typical congestive heart failure following alcoholic sprees. On the last admission he expired in 2 hours and 15 minutes.

Pertinent Autopsy Findings. The body measured 175 cm. and weighed 70 kg. The right pleural cavity contained 600 cc. of serous fluid and the left pleural cavity 300 cc. The heart weighed 500 gm. Externally, there were several nodulations in the right auriculoventricular groove (Fig. 3). On dissection, these proved to be a series of saccular aneurysms involving the right coronary artery (Fig. 4). The aortic ostium of the artery was of normal size. At a point 5 mm. from the ostium, the first of a series of saccular aneurysms was present. It measured 3.2 cm. in diameter and was filled with a brown-



Fig. 5. Cross section of aneurysm of right coronary artery showing thrombosis.

ish-tan thrombus (Fig. 5). There was central canalization of the thrombus. Continuing distally, there were 2 similar aneurysms separated by short sections of normal sized artery. All of the aneurysms had thin, calcific walls and contained thrombotic material. The left coronary artery showed a small aneurysm 1 cm. from its aortic orifice. There was no evidence of recent infarction of the myocardium. Numerous small grayish areas of fibrosis were present in the anterior and posterior walls of the left ventricle. The wall of the left ventricle was thinner than normal varying in thickness from 12 mm. at the base to 5 mm. at the apex. The right ventricular wall showed no evidence of old or recent infarction. It measured 4 mm. in thickness. The abdominal portion of the aorta was markedly sclerotic. There was a fusiform aneurysm 5 cm. in diameter just above the bifurcation. The walls of the aneurysm were thin and calcific areas were present.

The lungs were edematous and showed some emphysema. The kidneys weighed only 85 gm. each and microscopically showed nephrosclerotic changes.

The cranial cavity was not examined because of the limitation of the autopsy permission.

DISCUSSION

In the particular case reported here (Fig. 1 and 2) and confirmed at autopsy (Fig. 3, 4 and 5), large multiple aneurysms of the right coronary artery were revealed on routine roentgenograms of the chest as overlapping calcific rings of density closely associated with the right border of the heart and measuring several centimeters in diameter. There were several small ring calcifications involving the left coronary artery as well. Unfortunately, we were not sufficiently aware of this condition to make the proper diagnosis on the basis of roentgenographic findings prior to autopsy; in retrospect, the diagnosis should have been readily apparent.

Another point of considerable interest and importance is the association of aneurysm of the coronary vessels with abdominal aortic aneurysm. Daoud *et al.* state that in 8 of their 10 cases death was directly related to an abdominal aortic aneurysm which was not contiguously (by dissection or extension) associated with aneurysm of the coronary vessels. This fact is of considerable interest since our case at autopsy likewise revealed the presence of a small fusiform type of abdominal aortic aneurysm.

SUMMARY

Aneurysmal dilatation of the coronary rateries prior to the report of Daoud *et al.*[1] had been considered to be relatively uncommon. The finding by this group of 10 instances in 694 autopsied cases suggests that it is not so rare as previously supposed.

In the present case report, the aneurysms of the coronary vessels were clearly evident in conventional roentgenograms of the chest and diagnosis by an informed radiologist would have been possible.

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CALCIFICATION WITHIN CONGENITAL ANEURYSMS OF THE VEIN OF GALEN*

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ANEURYSMS of the vein of Galen are rare if use of the term is restricted to those vascular malformations in which a saccular vein of Galen is in direct communication with one or more large arteries, the internal venous system is inconspicuous and the aneurysm drains directly into the straight venous sinus. Excluded from consideration are vascular malformations draining entirely or in part into the deep venous system and in which the vein of Galen is enlarged as a venous tributary. In the latter instance, vessels, both capillary and venous, are interposed between the arterial component and the vein of Galen.

The literature contains 31 cases in which the diagnosis was established by angiographic studies or by postmortem examination (Table 1). Calcification within the wall of the aneurysm has been reported in 6 cases ranging in age from 15 to 34 years. The subject of the present report was 9 years of age when the incidental discovery of intracranial calcification prompted his admission to the hospital for diagnostic studies. This patient is the youngest of 7 patients in whom the diagnosis of aneurysm of the vein of Galen was possible on the basis of routine roentgenograms of the skull.

PREVIOUS REPORTS OF CALCIFIED ANEURYSMS

Alpers and Forster[1] reported the first example of roentgenographically visible calcification within an aneurysm of the vein of Galen in 1945. The patient, an 18 year old male, had complained of headaches since the age of 6 years. Destruction of the dorsum sellae and a concentric ring of calcification in the area normally occupied by the pineal gland were found on roentgenograms of the skull. Ventriculograms showed eccentric projection of a mass into an enlarged right lateral ventricle. Death followed 2 days after craniotomy and visualization of the aneurysm.

The second calcified aneurysm was reported by Oscherwitz and Davidoff[19] in 1947. Their patient, a female 27 years old, had complained of headaches for 9 months and blurred vision for 3 months. A complete circle, 3.5 cm. in diameter, was formed by the calcified aneurysm. A ventriculogram was normal. The aneurysm was opened and packed at the time of surgery, and disappearance of the patient's headaches was attributed to thrombosis produced within the aneurysm.

Wolfe and France[28] in 1949 reported a 21 year old female who had suffered from headaches for 15 years and who presented with ptosis, external squint and papilledema. A spherical calcification in the region of the pineal gland and ventriculographic demonstration of deformity of the aqueduct of Sylvius by a mass in this region resulted in a diagnosis of neoplasm. She died prior to a proposed shunting procedure, and an aneurysm of the vein of Galen was discovered at the time of postmortem examination.

The fourth case was one of the 2 patients described by Boldrey and Miller[2] in 1949. The aneurysm in this patient, an 18 year old male, was demonstrated by angiography and the midline calcification seen on plain roentgenograms conformed to the aneurysmal sac. A cranial bruit was diminished following ligation of the carotid artery. This case was the first in which the diagnosis of a calcified aneurysm was

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TABLE I

Authors	Case	Age	Sex	Clinical Findings					Treatment	Outcome
				Hydrocephalus	Cardiac	Bleeding	Seizures	Calcification		
Steinheil[24] (1895)	1	49 yr.	M				+			Death
Jaeger, Forbes and Dandy[13,14] (1937)	2	5 yr.	M	+					Carotid ligation	Death
Russell and Nevin[23] (1940)	3	17 mo.	M	+						Death
	4	17 mo.	M	+					Posterior fossa exploration	Death
Alpers and Forster[1] (1945)	5	18 yr.	M	+				+	Exploratory craniotomy	Death
Oscherwitz and Davidoff[19] (1947)	6	27 yr.	F					+	Craniotomy and packing of aneurysm	Survival
Lumsden[16] (1947)	7	7 mo.	M			+	+			Death
Boldrey and Miller[2] (1949)	8	16 mo.	M	+		+			Ligation of carotid artery; clipping of postcerebral artery	Survival
	9	18 yr.	M					+	Ligation of carotid artery	Survival
Cloward[5] (1949)	10	34 yr.	M					+		Death
Wolfe and France[28] (1949)	11	21 yr.	F	+			+	+	Cerebrospinal fluid shunt	Death
Pollock and Laslett[20] (1953)	12	4 da.	M	+	+					Death
Petit-Dutaillis, Guiot and Berdet[22] (1953)	13	4 mo.	M	+						Survival
Clement, Gerbeau, Combes-Hamille, Pertuiset and Petranca[4] (1954)	14	1 mo.	F		+					Death
	15	7 mo.	F	+	+					Death
French and Peyton[7] (1954)	16	21 mo.	M	+					Craniotomy; ligation of feeding arteries	Survival
Nayroc *et al.*[18] (1956)	17	43 yr.	M		+				Craniotomy; ligation of feeding arteries	Survival
Hirano and Terry[11] (1958)	18	28 mo.	F	+						Death
Thomson[26] (1959)	19	12 mo.	M	+						Survival
Gibson, Taylor and Richardson[9] (1959)	20	6 mo.	F	+					Cerebrospinal fluid shunt	Death
Taveras and Poser[25] (1959)	21	14 mo.	M	+						Survival?
	22	7 mo.	F	+	+				Craniotomy; ligation of feeding arteries	Survival
Claireaux and Newman[3] (1960)	23	4 da.	M		+		+			Death
	24	16 da.	M		+					Death
Poppen and Avman[21] (1960)	25	16 yr.	M	+		+		+	Craniotomy; excision of aneurysm	Survival
Gagnon and Boileau[8] (1960)	26	2 da.	M	+	+					Death
Hirano and Solomon[11] (1960)	27	6 wk.	M	+	+	+				Death

Table I (*continued*)

Authors	Case	Age	Sex	Clinical Findings					Treatment	Outcome
				Hydrocephalus	Cardiac	Bleeding	Seizures	Calcification		
Litvak, Yahr, and Ransohoff[15] (1960)	28	7 mo.	F	+	+				Craniotomy; excision of aneurysm	Survival
	29	9 mo.	F	+					Craniotomy; ligation of feeding arteries	Death
Glatt and Rowe[10] (1960)	30	3 da.	M		+					Death
Van Epps and Perret[27] (1963)	31	16 mo.	F	+			+		Craniotomy; ligation of feeding arteries	Survival
Wilson and Roy	32	9 yr.	M	+	+			+		Survival

established by means of angiography.

The fifth case was cited by Boldrey and Miller[2] in their 1949 paper. In a personal communication to the authors, Cloward[5] described a 34 year old male in whom a calcified aneurysm was verified by postmortem examination.

Poppen and Avman[21] reported the sixth calcified aneurysm in 1960. A crescentic area of calcification was discovered in the pineal region of a 16 year old male who was being investigated because of a disturbance in equilibrium, vertigo and blurred vision. The aneurysm was totally removed after the diagnosis was established by bilateral carotid and left vertebral angiography. A diagnosis of hydrocephalus was made on the basis of angiographic findings.

A report of the seventh case follows.

REPORT OF A CASE

A. F. (L-53-90677), a 9 year old male, was seen on an annual visit to the Neurosurgery Clinic in June, 1962, and skull roentgenograms were taken as a matter of interest. A thin curvilinear calcific density was seen in the region of the pineal gland on the lateral roentgenogram (Fig. 1). The calcification was not clearly visible in frontal projection. His parents were advised by letter that he should enter the hospital for evaluation.

He was admitted to the Charity Hospital on August 10, 1962. The only complaint given by the patient was that of infrequent headaches. His mother considered him normal although he was not as active as his classmates. His head was first thought to be abnormally large at the age of 7 months. His motor and language development had been normal, although at the age of 13 months he was called a case of "arrested hydrocephalus" by a resident in Pediatrics.

He had developed frequent, recurrent and severe nose bleeds beginning at the age of 2 years for which he was hospitalized on the Ear, Nose and Throat Service for evaluation at the age of 3 years. A soft vascular mass overlying the bridge of his nose was described, but no source of bleeding was discovered within the nasal cavity. Recurrent nose bleeds continued until the age of 4 years. At 2 years of age, he was fitted with shoes for the correction of bilateral pronation deformity and pes planus.

Family History. The patient was one of 8 healthy children, and similar or related disorders had not occurred in the family of either parent.

Physical Examination. The patient was a slender boy with a large head, the circumference measuring 65 cm. The blood pressure was 114/72, pulse 84 and temperature 98.6° F. His speech was halting and had a nasal quality. There were prominent veins over the forehead and scalp, the most conspicuous of which was a fluctuant blue mass, 1 cm. in area, overlying the nasofrontal junction.

The cranial nerves were normal. His station and gait were normal although he had slight difficulty with tandem walking. Muscle strength was fair. Coordination was mildly impaired and there was a terminal tremor on finger to nose

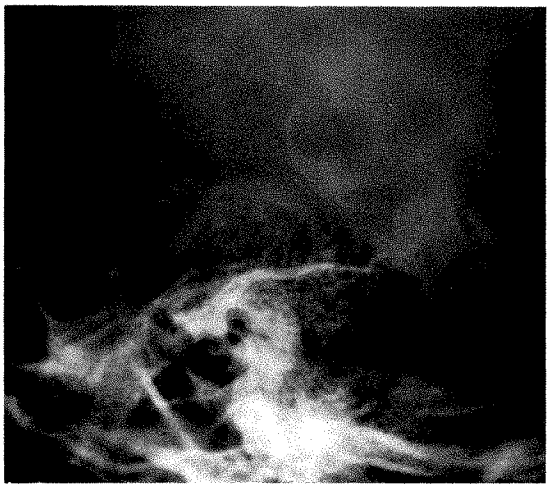


FIG. 1. Plain lateral roentgenogram showing linear calcification within the wall of the aneurysm.

testing and awkwardness of rapid alternating movement on the left. Deep and superficial reflexes were normal. The final impression was that the patient was awkward for his age.

He was tested by the Department of Clinical Psychology. His intellectual functioning on the verbal level was above average. He had difficulty with visual motor coordination which interfered with reading and writing skills. A slightly below normal social-emotional development was attributed to an overly attentive and protective attitude of the parents. He was tense and was thought to be incapable of coping with failure or threat of failure. His full intelligence quotient was 100.

Electroencephalography. An electroencephalogram on August 23, 1962 showed a background alpha rhythm of 10/sec. which was often obscured by the predominant slowing seen throughout the record; 4–6/sec. slowing was scattered fairly symmetrically through the record. At times the right or left hemisphere would be prominently slowed. Occasional 2–3/sec., 100 m.v. slowing was seen in the record. When the patient drowsed and slept, central and parietal slowing of 150 m.v. and 3/sec. was seen. Central transients were frequent. Symmetric bursts of 4–5/sec., 200–250 m.v. slowing were seen during sleep and drowsiness. Impression: abnormal electroencephalogram, generally slow with occasional symmetric bursts.

Cardiac Evaluation. He was seen in consultation by the Pediatric Cardiology Service for the purpose of cardiac evaluation. A continuous murmur was described, loudest just below and in front of the right mastoid process, but which could be heard faintly over the temporal regions and the left mastoid process. The murmur was transmitted bilaterally downward to the clavicles. The heart tones were good. A Grade I–II systolic murmur was heard in the pulmonic area. A roentgenogram of the chest indicated early left ventricular enlargement. An electrocardiogram was normal. Cardiac catheterization was done with the finding of a relatively high oxygen content in the superior vena cava and a small arteriovenous oxygen difference in samples removed from the superior vena caval branches. This was interpreted as an indication of a high flow rate in the upper part of the body. Pressure relationships were normal. Based on the measured oxygen consumption of 156 cc./min., the cardiac output was calculated as 6.24 l./min. Surface area was estimated as 1.2 m.^{2}. The cardiac index was therefore 5.2 l./m.^{2}/min.

Laboratory Findings. A lumbar puncture was normal. Routine hemogram and urinalysis were also normal. An EPA of the chest was reported as normal except for the cardiac enlargement mentioned previously.

Pneumoencephalography. A total of 16 cc. of air was injected on August 14, 1962, using local anesthesia. The rostral portion of the fourth ventricle and aqueduct were deformed and displaced towards the clivus in a smooth curve (Fig. 2). The lateral ventricles were enlarged.



FIG. 2. Pneumoencephalogram showing air in the aqueduct of Sylvius and fourth ventricle which are bowed anteriorly.

Angiography. Right carotid angiography was performed on August 16, 1962, left carotid angiography on August 21, 1962, and percutaneous right vertebral angiography on August 24, 1962.

Right carotid injection filled a saccular aneurysm of the vein of Galen through two branches of the middle cerebral artery (Fig. 3). The posterior communicating, anterior choroidal and anterior cerebral arteries did not opacify. The aneurysm drained directly into a greatly enlarged straight sinus and thence into both transverse sinuses (Fig. 4).

Left carotid injection filled the aneurysm by way of the right pericallosal artery (Fig. 5).

Right vertebral injection filled the aneurysm through both posterior cerebral arteries (Fig. 6, *A* and *B*). The vertebral artery was dilated and the basilar artery formed a large loop to the left of the midline. Several varicosities were seen along the course of small veins leading away from the aneurysm.

Follow-up. The patient was discharged from the hospital on August 26, 1962. He has been seen at intervals of 3 months and on his last clinic visit in March, 1963, his state of health was unchanged.

ROENTGENOGRAPHIC FINDINGS

Plain roentgenograms of the skull may give evidence of increased intracranial pressure. Deepening of the grooves of the transverse sinuses and enlargement of emissary vein foramina and grooves may be found normally, although they are particularly prominent in the presence of intracranial hypertension from whatever cause. These findings are therefore of little value as an indication of pathologically enlarged venous channels.

Visible calcification within the wall of an aneurysm of the vein of Galen is virtually pathognomonic if the following criteria are satisfied: (1) the calcific deposit is thin and delicate, and forms a complete or in-

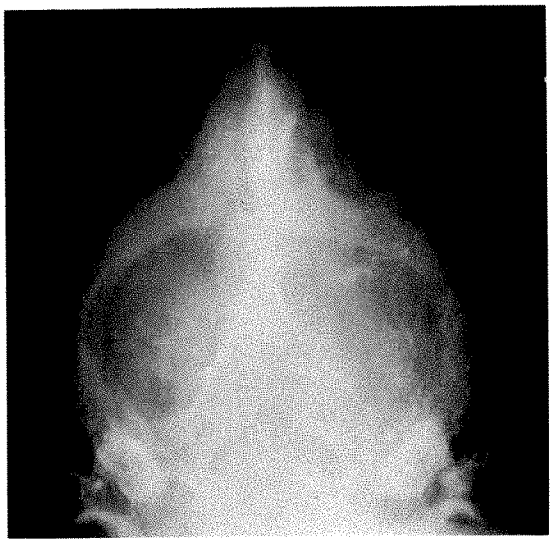


Fig. 4. Venous phase showing enlarged transverse sinuses bilaterally.



Fig. 3. Right carotid angiogram showing 2 branches of the middle cerebral artery passing directly into the aneurysm.

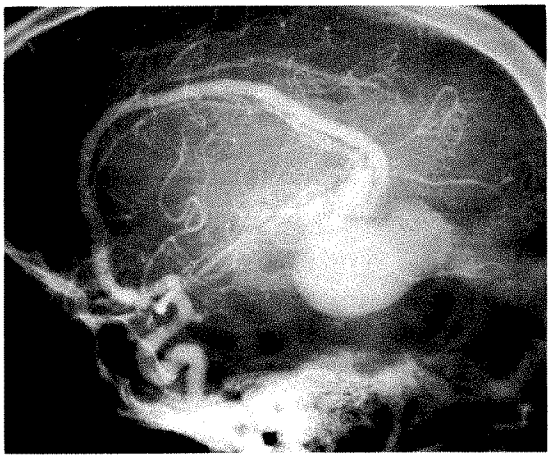


Fig. 5. Left carotid angiogram showing filling of the aneurysm almost entirely by way of the right pericallosal artery.

complete ring; (2) it is situated in the region normally occupied by the pineal gland in the lateral projection and very near or in the midline in the frontal projection; and (3) the ring of calcification, whether complete or incomplete and constructed by projecting imaginary lines, is large, exceeding 2.5 cm. in diameter.

A diagnosis based soley upon plain roentgenograms should be made only in an older child or adult since the youngest patient with calcification yet reported was 9 years of age. Pineal tumors do not calcify in this manner.[17] Epidermoids in the quadrigeminal area are rare and could closely mimic an aneurysm. A lipoma should be differentiated by a central area of radiolucency.

Air contrast studies have been performed in patients with this condition during infancy because of progressive hydrocephalus or in later life because of increased intracranial pressure and a presumptive diagnosis of brain tumor. Large aneurysms may deform or project into the posterior portion of one or both lateral ventricles. An aneurysm may behave like a pineal tumor by compressing the quadrigeminal plate and the subjacent aqueduct of Sylvius. The larger aneurysms may deform the rostral portion of the fourth ventricle. Significant narrowing of the aqueduct leads to dilatation of the lateral and third ventricles. Preservation of a relatively normal configuration of the third ventricle is uncommon with pineal neoplasms and should alert the examiner to the possibility of an aneurysm of the vein of Galen or other extrapineal mass.

Angiography is the definitive diagnostic procedure. One major artery, most commonly the posterior cerebral, may constitute the sole source of arterial blood, or several arteries may be involved. Complete delineation of the malformation requires that the carotid arteries be visualized on each side as well as the vertebral-basilar circulation. Bilateral vertebral artery injec-

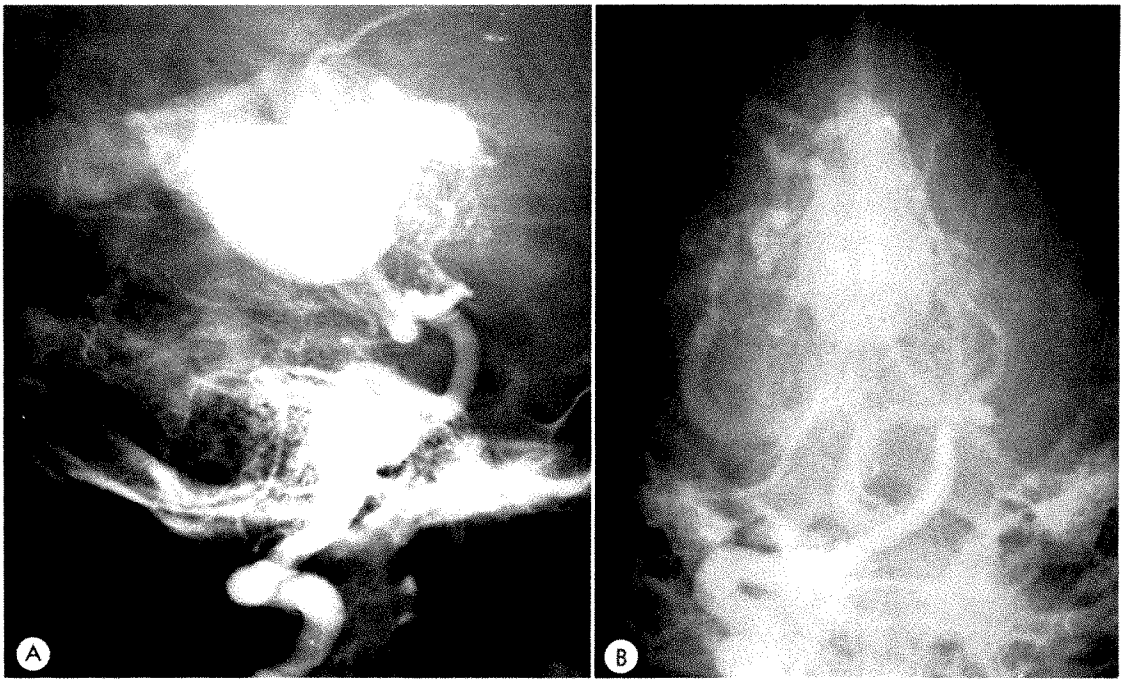


FIG. 6. (*A* and *B*) Right vertebral angiograms showing that the aneurysm is supplied by both posterior cerebral arteries. The vertebral and basilar arteries are dilated and the latter describes a wide loop to the left of the midline.

tion is unnecessary, judging from the cases described in the literature. A separate arterial component was demonstrated by injection of each of the 3 vessels studied in the case reported above. Arterial blood flows directly through a much enlarged straight sinus into the transverse sinuses in a typical case. Smaller venous structures collateral to the aneurysm may be visualized. These latter vessels have thin walls which may rupture due to their high intravascular pressure.

CLINICAL FEATURES

There are basically 3 mechanisms of symptom production: (1) the aneurysm may behave as a mass in the manner of a pineal tumor to produce obstructive hydrocephalus and compression of the midbrain; (2) bleeding may result from the rupture of venous tributaries and repeated subarachnoid hemorrhage may lead to the development of hydrocephalus; and (3) large arteriovenous shunts may impose a strain on cardiac function during infancy.[12] Twenty-one of the 32 cases under consideration were less than 2 years of age. Thirteen were below 1 year of age at the time of diagnosis and 10 of this number had cardiac manifestations of a systemic arteriovenous shunt. It is notable that males have predominated in a ratio of 22/10.

The role of chronic cerebral ischemia in the production of clinical manifestations is less clear. Angiographic evidence of diversion of blood from major cerebral arteries into the arteriovenous shunt suggests that some degree of cerebral ischemia might exist. This could account for epilepsy, mental retardation and other neurologic abnormalities which have been reported in this condition in the absence of hydrocephalus.

TREATMENT

Hydrocephalus may be treated apart from the vascular malformation. A procedure designed only to relieve hydrocephalus may be indicated in a recognized case, and it is likely that such procedures have been performed in a considerable number of cases in which the vascular malformation was unsuspected.

A direct surgical attack upon the aneurysm has been reported in 9 cases, only 1 of which died as a result of the operation.[7,15,16,18,19,21,25,27] Craniotomy and interruption of the arterial component of the fistulous communication, alone or with excision of the aneurysmal sac, requires complete preoperative angiographic study. Encouraged by these successful reports, it seems likely that neurosurgeons will undertake to excise the more favorable of these malformations in the future.

SUMMARY

Thirty-one cases of aneurysm of the vein of Galen had been reported previously. Calcification within the wall of the aneurysm was visible on plain skull roentgenograms in 6 of this number. The present communication describes an additional case in which calcification of the aneurysm was discovered at the age of 9 years, the youngest patient heretofore reported.

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SPONTANEOUS THROMBOSIS OF INTRACRANIAL ANEURYSMS*

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THE increased use of cerebral angiography in the last two decades has made it possible to diagnose cerebrovascular diseases more correctly, thus allowing the physician to provide an immediate and more specific course of treatment.

Aneurysms which are demonstrated by cerebral angiography present no particular diagnostic problem. When they are completely thrombosed, however, they are not demonstrable in the arteriogram because the injected contrast substance cannot enter the aneurysmal sac. Similar difficulties are encountered in the presence of partial thrombosis in which case only the patent part of the aneurysm is visible, thus deceiving the interpreter as to its true size. It is mainly the last group with which we are here concerned.

In many instances the method of treatment depends upon accurate information regarding the actual size of the aneurysm and the presence of thrombosis. For this reason a series of cases was reviewed in an effort to find signs which would prove helpful in establishing the roentgenologic diagnosis of thrombus in intracranial aneurysms.

REVIEW OF THE LITERATURE

Complete thrombosis occurring spontaneously is rare and only a few cases have been reported in the medical literature. Krayenbühl[9] found in an extensive series of 7,452 autopsies only 1 completely "fibrosed" aneurysm. The first case of an entirely calcified aneurysm originating from the internal carotid artery was described in 1875.[5] Subsequent cases with postmortem findings of completely clotted and partly calcified aneurysms of the circle of Willis,[11] and of the middle[8] and the anterior fossae[14] were reported.

The first recognition of an aneurysm in the plain skull roentgenogram dates back to 1916.[4] Ten years later a comprehensive review of aneurysms of the circle of Willis and of the internal carotid artery was published, with attention focused on the calcifications of the wall and the erosion of the adjacent bone structures.[16] In the following years more detailed accounts concerning the bone changes, which included unilateral erosion of the sella turcica, enlargement of the optic foramen, the supraorbital fissure and the foramen lacerum and elevation of the sphenoid wing and anterior clinoids[7,12] appeared in the literature. These findings were principally associated with cavernous sinus aneurysms. According to one study,[2] the incidence of aneurysmal wall calcification is 6 per cent.

With the advent of arteriography, the diagnosis of aneurysms was more easily made; however, the recognition of thrombosis remained difficult. The first demonstration of a thrombus was achieved by injecting lipiodol into the sphenoid sinus which delineated a filling defect produced by an intracavernous sinus aneurysm. Subsequent arteriograms showed an area not outlined by contrast substance situated between the aneurysm and the sphenoid sinus; thus the actual size of the thrombus was revealed.[15] The visualization of flecks of contrast medium around an aneurysm has been suggested as indicating contrast material within a thrombus.[9] Various signs for the determination of complete thrombosis have been described, among which is the

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Table I

DISTRIBUTION OF INTRACRANIAL ANEURYSMS

Location	No. of Cases	Per Cent
Internal carotid artery	27	24.5
Posterior communicating artery	18	16.3
Anterior cerebral artery	14	12.7
Middle cerebral artery	29	26.3
Anterior communicating artery	16	14.5
Unknown origin	3	2.7
Basilar artery	3	2.7
Total	110	

nonfilling of the aneurysm and its distal artery associated with tapering of the related proximal artery.[3,9] Pneumoencephalography has been credited with revealing a filling defect which, in conjunction with arteriographic findings, implied the presence of a completely thrombosed aneurysm.[3]

METHOD AND RESULTS

In our efforts to determine whether thrombosis of intracranial aneurysms could be diagnosed by arteriography, the initial task was to review the autopsy and histology files of the Department of Neuropathology. One hundred and ten suitable cases were found. This number represents material collected over a period of 10 years, from 1952 through 1961. The majority of the cases were seen during the last 5 years owing to the increased admission of patients afflicted with cerebrovascular disease. The ages ranged from 15 years to 78 years. The sex distribution indicated a slight predominance of females: 50 were males and 60 were females.

The aneurysms occurred at various locations as shown in Table I. Cases in which the aneurysm originated from the middle cerebral artery comprised the largest group (29 or 26.3 per cent); those in which the aneurysm arose from the internal carotid artery and included the cases at the bifurcation and the junctions of the internal carotid, anterior and middle cerebral artery were next in number (27 or 24.5 per cent). There were 18 cases (16.3 per cent) of aneurysm from and about the posterior communicating and internal carotid artery; 16 cases (14.5 per cent) of aneurysm originating from the anterior communicating artery and 14 cases (12.7 per cent) of aneurysm from the anterior cerebral artery. Only 3 patients (2.7 per cent) had aneurysm at the basilar artery and the same number had aneurysm of unknown origin.

The size of each aneurysm was recorded and its length was used for classification. Descriptive terms of sizes are usually very individual and often lead to erroneous conceptions. The aneurysms were therefore arbitrarily divided into 4 categories: (1) the small-sized group (measuring from 1 to 5 mm.) with a total of 44 cases; (2) the medium-sized group (measuring from 6 to 15 mm.) consisting of 38 cases; (3) the large-sized group (ranging from 16 to 30 mm.) comprising 14 cases; and (4) the very large-sized group (31 mm. and over) with 5 cases; 9 aneurysms were of undetermined size. Spontaneous thrombosis was noted in 26 instances: 8 in Group 1; 11 in Group 2; 5 in Group 3; and 2 in Group 4 (Table II).

Minute thrombi are not visible in the arteriogram unless they produce some irregularity at the wall of the aneurysm. The thrombi referred to in this series are those which have at least obliterated a fifth of the lumen. The thrombi were of varying sizes and located principally in the fundus.

The histologic reports of arteries adjacent to the aneurysm were reviewed and showed that partial arterial thrombosis was present in 18 cases. Atheromatosis was present in 61 cases at various sites of the arterial tree but mainly in the vicinity of the aneurysm. No pathologic changes were found in the remaining 31 arterial specimens. A correlation of the findings in the anatomicopathologic specimens with the corresponding arteriograms was attempted. It was found, however, that in only 62 of the 110 cases was arteriography performed and 48 patients did not undergo arteriography. In the group without arteriography, 12 an-

Table II

SEX DISTRIBUTION AND SIZE OF INTRACRANIAL ANEURYSMS

Sex		Group 1 Small Size (1–5 mm.)	Group 2 Medium Size (6–15 mm.)	Group 3 Large Size (16–30 mm.)	Group 4 Extra Large Size (31 mm.–over)	Undetermined Size	Total
M	F						
Autopsy and Surgical File							
50	60	44	38	14	5	9	110
Roentgenographic File (Intracavernous sinus aneurysms)							
2	10	0	0	6	6	0	12
Grand Total							
52	70	44	38	20	11	9	122
Thrombosed Aneurysms							
		8	11	5	2	0	26

eurysms contained thrombi which most likely would have been demonstrated on arteriograms. The remaining 36 cases showed no evidence of thrombosis macroscopically. Of the 62 patients with arteriography, no thrombosis was found in 45 cases; the remaining 17 cases contained thrombi of varying extent (Table III). Each of the pathologically verified cases of aneurysms was carefully correlated with the carotid angiograms. All roentgenograms of each case, including those of various time phases, were reviewed.

Study of the arteriograms of 17 cases of aneurysms proven by autopsy or surgery showed 3 which were unsatisfactory for evaluation from the roentgenologic point of view. Due to the rather small number of proven aneurysms available, 9 cases of carotid cavernous sinus aneurysms were added to the series, all of which had definite roentgenologic proof of partial thrombosis and could, therefore, logically be included. The actual number of cases of carotid cavernous sinus aneurysms in which arteriography was performed was 14; 2 had been verified pathologically and were included in the main series of proven cases; 3 cases showed no evidence of thrombosis, thus, only the remaining 9 cases were added. In this group of patients the major-

Table III

INCIDENCE OF THROMBOSIS OF INTRACRANIAL ANEURYSMS

110 Aneurysms (Autopsy and Surgical File)		
Arteriography—Thrombosis	17*	62
Arteriography—No Thrombosis	45	
No Arteriography—Thrombosis	12	48
No Arteriography—No Thrombosis	36	
110 Arterial Specimens		
Arterial Thrombosis	18	
Atheromatous Changes	61	
No Pathologic Findings	31	
Intracavernous Sinus Aneurysms (Roentgen-Ray File)		
Arteriography—Thrombosis	9	12
Arteriography—No Thrombosis	3†	
Total		122

* In 3 cases the arteriograms were unsatisfactory for evaluation.

† These 3 cases are not included in the series.

TABLE IV

ROENTGEN SIGNS IN THE PRESENCE OF THROMBOSIS OF INTRACRANIAL ANEURYSMS

Partial Thrombosis	No. of Cases	Displacement	
		With	Without
No associated changes	7	7	0
Bone changes	3	3	0
Calcification	3	2	1
Bone changes plus calcification	1	1	0
Semilunar defect	2	0	2
Central defect	1	0	1
Contrast lag	3	0	3
Meningioma simulating aneurysm	1	1	0
Complete thrombosis	2	1	1
Total	23	15	8

ity were females. Of the 12 cases of cavernous sinus aneurysms, 6 could be classified into Group 3 and 6 into Group 4 (Table II). The ages ranged within the limits of those in the main series. Altogether 23 cases, 14 histologically verified and 9 roentgenologically proven, were available for review.

In Table IV are listed the roentgen signs which were noted in the presence of partial and complete aneurysmal thrombosis. Displacement of the vessels adjacent to the thrombosed aneurysm was found in 15 cases, mainly in the larger type of aneurysm; 7 cases showed arterial displacement only, without secondary changes. The remaining 8 cases were associated with other changes, such as erosion or bone destruction, aneurysmal wall calcifications or bone changes and calcifications combined, a meningioma simulating aneurysm, and 1 case with a large aneurysm showing complete thrombosis. No vascular displacement was noted in 8 cases, with findings of a semilunar filling defect, central clot, lag of contrast material and complete thrombosis of the aneurysm.

In 7 cases there was displacement of the intracranial vessels only, outlining in the majority the thrombosed part of the adjacent aneurysm without evidence of secondary changes. Displacement of the carotid artery in the presence of cavernous sinus aneurysms associated with bone changes such as unilateral erosion of the sella turcica, widening of the superior orbital fissure and erosion of the lower, outer wall of the optic foramen, was noted in 3 cases.

ILLUSTRATIVE CASES

Displacement associated with bone changes and aneurysmal calcifications was present in 1 case (Case 1).

CASE 1. A male, aged 67, was admitted in 1955 with a 7 year history of ptosis, proptosis and impairment of vision of the right side. For about the same length of time he complained of weakness of both hands. Nine months prior to admission his mental capacities gradually began decreasing and in the last 10 days he became confused and disoriented.

On examination, the patient was found to have complete ptosis and proptosis as well as complete ophthalmoplegia on the right side. Marked weakness and wasting of the forearms and particularly of the triceps muscles were noted, predominantly on the left.

The plain skull roentgenograms revealed gross destruction of the pituitary fossa (Fig. 1*A*). The posterior clinoids were thinned and the floor of the fossa was grossly excavated and depressed. There was evidence of a shell-like calcification at the posterior aspect of the sella turcica. The frontal study showed evidence of erosion of the right supraorbital fissure. Carotid angiograms demonstrated an aneurysm measuring 1×1.8×1.5 cm. located in the cavernous sinus. It was presumed that the aneurysmal sac was thrombosed to a considerable extent, thus explaining the widening of the siphon, posterior displacement of the cavernous portion of the carotid between lingula-petrosal process and siphon loop, erosion of bone and curvilinear calcification (Fig. 1*B*).

At autopsy, a large aneurysm was found at the intracavernous portion of the carotid artery. Anteriorly and posteriorly it had eroded bone. A moderate sized thrombus was present within the aneurysm.

Calcifications associated with aneurysms and displacement of one of the adjacent

intracranial arterial branches were found in 2 instances. Only a portion of the aneurysm was outlined by contrast material. In these instances the actual size of the aneurysm can be determined by the calcifications away from the aneurysm and the extent of the displacement of the vessels (Case II).

Case II. A female, aged 67 years, was admitted in 1959 because a brain tumor was suspected. Two years prior to admission she had a stroke resulting in right hemiparesis. She had been bedridden ever since then with varying degrees of consciousness.

On examination she was dysarthric and incontinent. The cerebrospinal fluid was xanthochromic with a protein of 180 mg. per cent. The patient gradually deteriorated, became comatose and did not respond to painful stimuli. The pupils were fixed and dilated and she developed bilateral papilledema. The plain skull roentgenogram showed a calcified lesion near the midline. A left carotid angiogram revealed a very large aneurysm extending upwards and backwards from the bifurcation of the left internal carotid artery. The anterior cerebral vessels were displaced about 2.5 cm. to the right. Intracranial calcifications close to the aneurysm suggested that the aneurysm was considerably larger (Fig. 2, *A* and *B*).

The patient died approximately 6 weeks after arteriography. Postmortem examination revealed a very large aneurysm, measuring 5×4 ×5.5 cm., originating from the internal carotid artery slightly above the posterior communicating artery. In the midzone of the aneurysm, a laminated clot was found occupying the lateral two-thirds of the posterior part of the aneurysm. A layer of laminated clot lined the ventral parts. A calcified mass was present in the posterior part of the aneurysm.

An aneurysm showing calcification but no vascular displacement was found in 1 case. A semilunar filling defect in an aneurysm was demonstrated in 2 cases. In 1 case the convexity of the defect pointed toward the contrast shadow and in the other away from it. At autopsy, thrombus was found in the fundal portion of the aneurysm (Case III).

Case III. A female, aged 52 years, was admitted in 1957 because of sudden collapse. About 4 years prior to admission she became unconscious for a short time and developed a right third nerve palsy and a left hemiparesis. This was followed by hypertension.

On examination there was evidence of complete right ptosis, a dilated right pupil and upward deviation of the right eye. Bilateral extensor plantar responses with absent abdominal reflexes were found. The blood pressure was 260

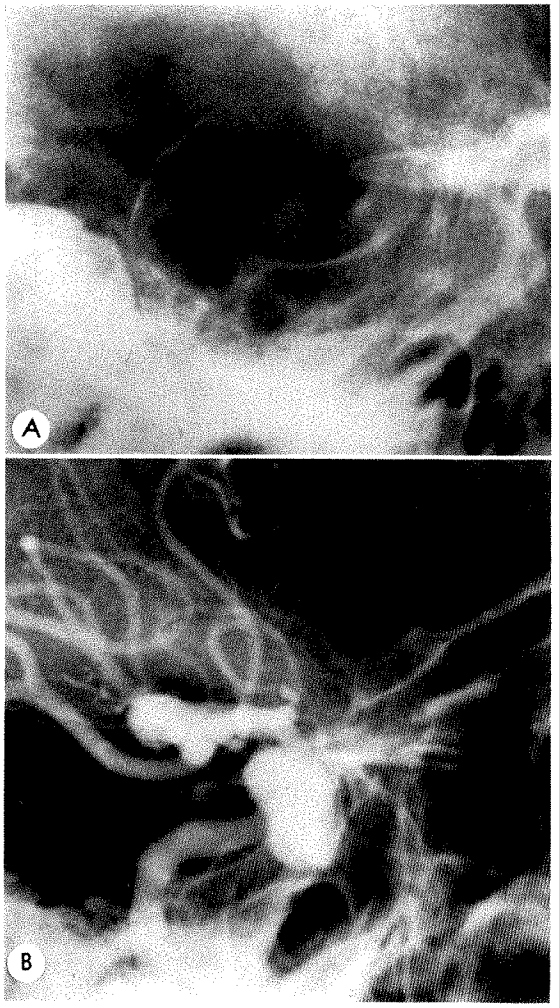


Fig. 1. Case I. (*A*) Lateral roentgenogram of the sella turcica showing a shell like calcification 1 cm. posterior to the dorsum sellae. There is gross destruction of the pituitary fossa. (*B*) Lateral carotid arteriogram demonstrates an intracavernous sinus aneurysm with widening of the siphon and posterior displacement of the carotid between lingula-petrosal process and siphon loop. The thrombus is delineated between the posterior aspect of the aneurysm and the shell like calcification.

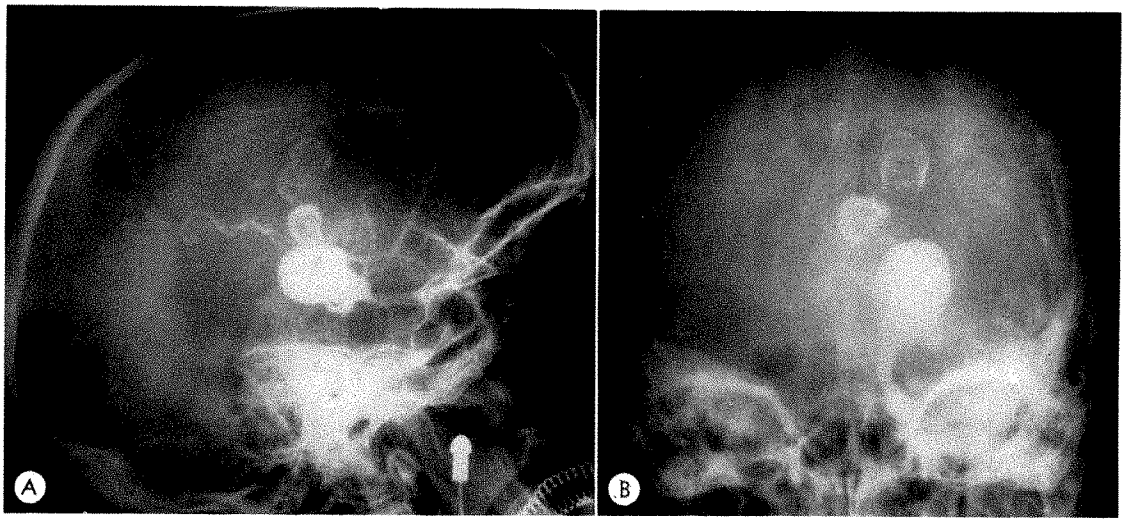


Fig. 2. Case II. (*A* and *B*) Carotid arteriograms showing a large aneurysm originating from the internal carotid artery. Calcification and displacement of the arteries delineate the true size of the partially thrombosed aneurysm.

mm. Hg and the cerebrospinal fluid pressure was 150 mm. A right carotid angiogram demonstrated an aneurysm originating from the right internal carotid artery at the origin of the posterior communicating artery (Fig. 3). The patient died 2 months after arteriography. Postmortem examination showed an aneurysm originating from the posterior communicating artery adjacent to the sella turcica with a thrombus in the fundal region.

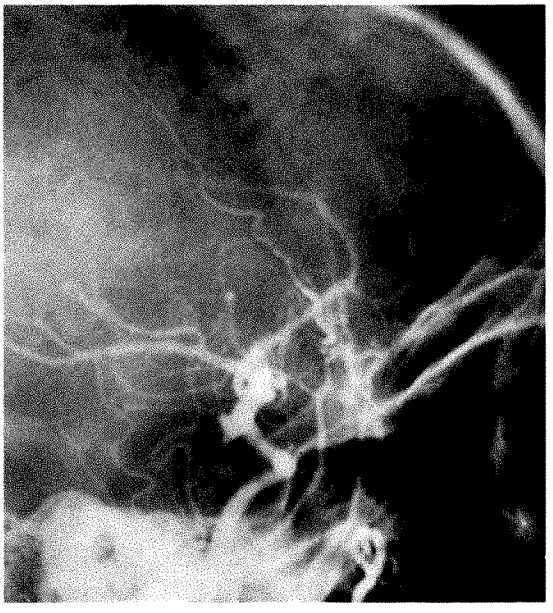


Fig. 3. Case III. Lateral carotid angiogram shows an aneurysm arising from the posterior communicating artery. A semilunar filling defect is seen at the posterior aspect of the aneurysm produced by thrombus located at the fundus.

In 1 instance a filling defect representing thrombus was found in the central portion of a readily demonstrable aneurysm. The diagnosis offered no particular difficulty (Case IV).

Case IV. A female, aged 55 years, was admitted in 1955. The patient had been well until 11 days prior to admission when she suddenly became unconscious for a short period of time and later complained of occipital headaches and pain throughout her body.

On examination neck stiffness with a negative Kernig sign was noted. Lumbar puncture revealed heavily blood stained fluid and a protein content of 160 mg. per cent. She then developed right hemiplegia and minimal dysphasia. Subsequently, the pupils became fixed and dilated and the eyes deviated to the left. The corneal reflex was absent on the right. The patient developed facial palsy, flaccid paralysis of the right arm and leg and minimal response to painful stimuli in the left arm and leg. There was flexor plantar response on the left with absent reflex on the right.

Left carotid angiography revealed a moderate sized aneurysm at the bifurcation of the internal carotid artery. An irregular negative filling defect was seen in the center of the aneu-

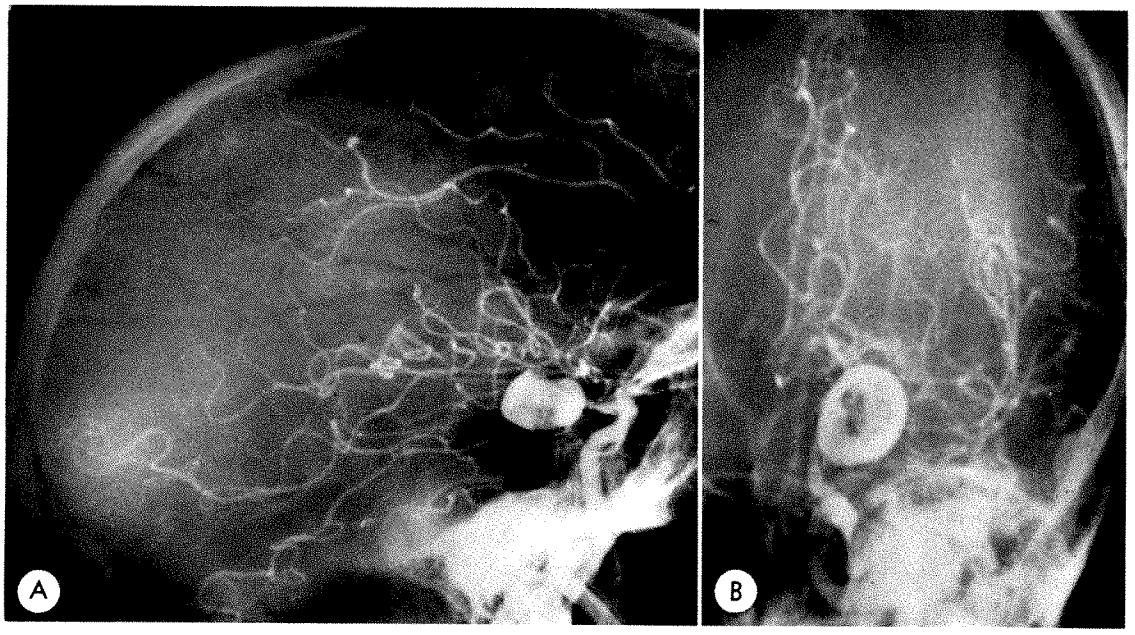


Fig. 4. Case IV. (*A* and *B*) Carotid arteriograms show aneurysm at the bifurcation of the internal carotid artery. An irregular central filling defect represents the aneurysmal thrombus.

rysm indicating the presence of thrombus (Fig. 4, *A* and *B*). The patient died a few months after carotid ligation. On postmortem examination an aneurysm was found just above the origin of the left posterior communicating artery. The left common carotid artery was ligated below its bifurcation. Recent thrombus filled the entire artery above and below the ligature and extended up and into the aneurysm.

In 3 cases retention of contrast material occurred in all phases, with contrast layering being demonstrated in 2 of these (Case V).

Case V. A male, aged 45 years, was admitted in 1961. At midnight of the day before admission the patient suddenly became rigid, drowsy and vomited at quarter hourly intervals. Although he improved slightly in the morning, he complained of headaches and vomited at about hourly intervals during the day. During the afternoon he experienced profuse sweating and chills for approximately an hour. Later he became progressively confused and disorientated.

On examination he had mild neck stiffness. Both plantar reflexes were extensor. The remainder of the examination was normal. The lumbar puncture showed markedly blood stained fluid.

Follow-up examination 12 hours later revealed increased neck stiffness, no light reaction of the left pupil, and mild right-sided weakness.

A left carotid arteriogram showed a large ragged, roughly spherical shaped aneurysm about 2.5 cm. in diameter taking origin from the termination of the carotid artery at its bifurcation. There was a considerable lag of contrast material in the aneurysm which was still opacified in the late phlebograms (Fig. 5, *A*, *B* and *C*). A right carotid angiogram showed a 1.2 cm. aneurysm at the bifurcation of the right internal carotid artery. The patient gradually deteriorated and died 3 days after admission.

Postmortem examination revealed a spherical aneurysm, measuring 1.2 cm., originating from the right internal carotid artery at its bifurcation. This aneurysm had not ruptured. A larger aneurysm, measuring 4 cm. in its largest diameter, was seen arising from the internal carotid artery at its bifurcation. The wall of the aneurysm was lined by laminated old thrombus to a depth of 1 cm.

In 1 instance the presenting aneurysm closely resembled a meningioma. The fine network of contrast material delineating a spongy thrombus simulated meningioma vessels. In such cases a careful evaluation

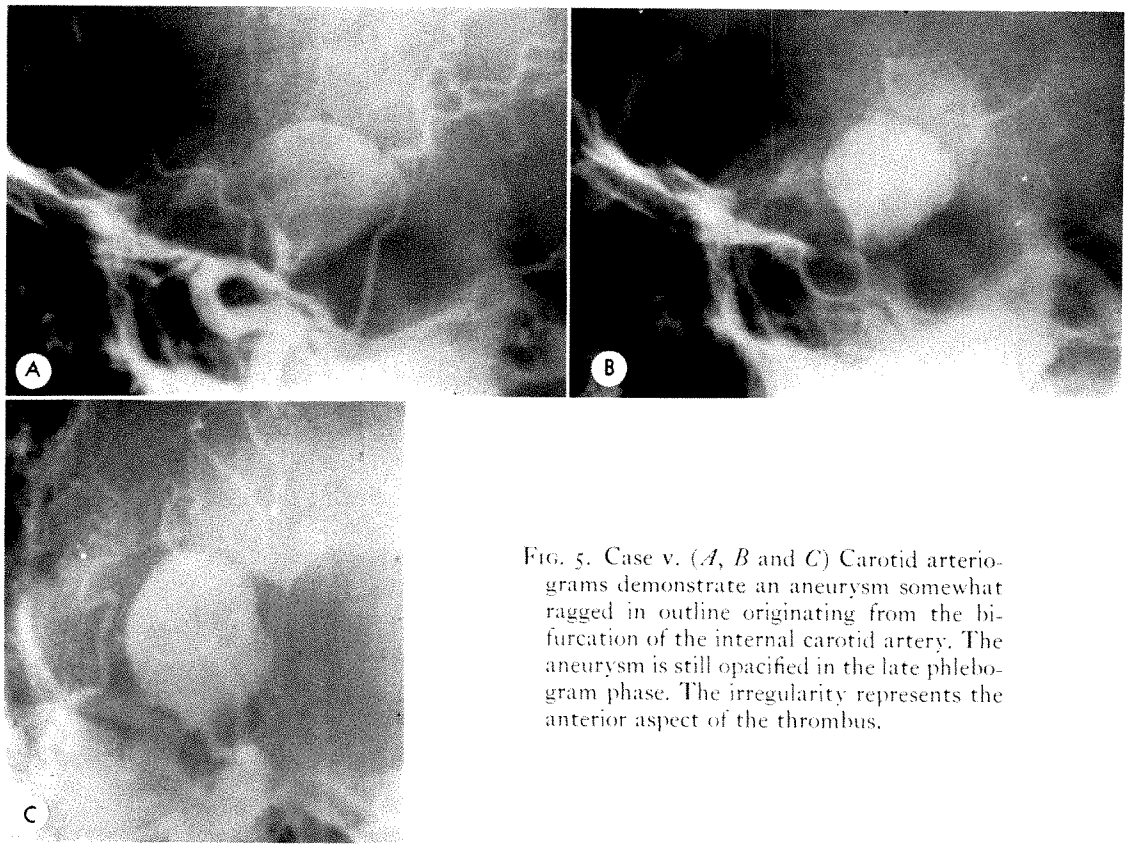


FIG. 5. Case v. (*A*, *B* and *C*) Carotid arteriograms demonstrate an aneurysm somewhat ragged in outline originating from the bifurcation of the internal carotid artery. The aneurysm is still opacified in the late phlebogram phase. The irregularity represents the anterior aspect of the thrombus.

of the roentgenograms will provide the correct diagnosis (Case VI).

CASE VI. A female, aged 52 years, entered the hospital because of double vision. Twenty-seven years prior to admission, the patient had complained intermittently of black spots in the central vision of both eyes followed by severe frontal headaches with occasional vomiting. Between the ages of 30 to 50 years the symptoms became less frequent. Eventually, double vision developed when looking to the right, with a recurrence 6 months before admission. Her vision gradually deteriorated.

On examination a mild visual impairment was found. There was optic atrophy, fourth and sixth nerve palsy, possible Horner's syndrome and a questionable involvement of the third nerve on the right.

A right carotid angiogram showed a well circumscribed collection of "pathologic vessels" in the middle fossa displacing the terminal portion of the carotid siphon anteriorly and elevating the middle cerebral arteries. This had the appearance of an extradural tumor (Fig. 6, *A*, *B* and *C*).

At operation a large bluish extradural tumor arising from the floor of the middle fossa was exposed measuring 4 cm. in diameter. Palpation gave the impression of the presence of a cystic lesion. Its appearance was that of an aneurysm. A needle was inserted through a firm capsule and pure arterial blood was aspirated.

In 2 instances complete thrombosis was present without contrast medium filling the aneurysm. In 1 case the aneurysm was only 2 mm. in size and no displacement of neighboring vessels was evident. In the other, displacement of the adjacent vessels was demonstrated along with certain features which resembled those of an avascular tumor such as a meningioma (Case VII).

CASE VII. A male, aged 62 years, was admitted because of headaches and personality changes which developed 6 months earlier. On examination there were signs of mental re-

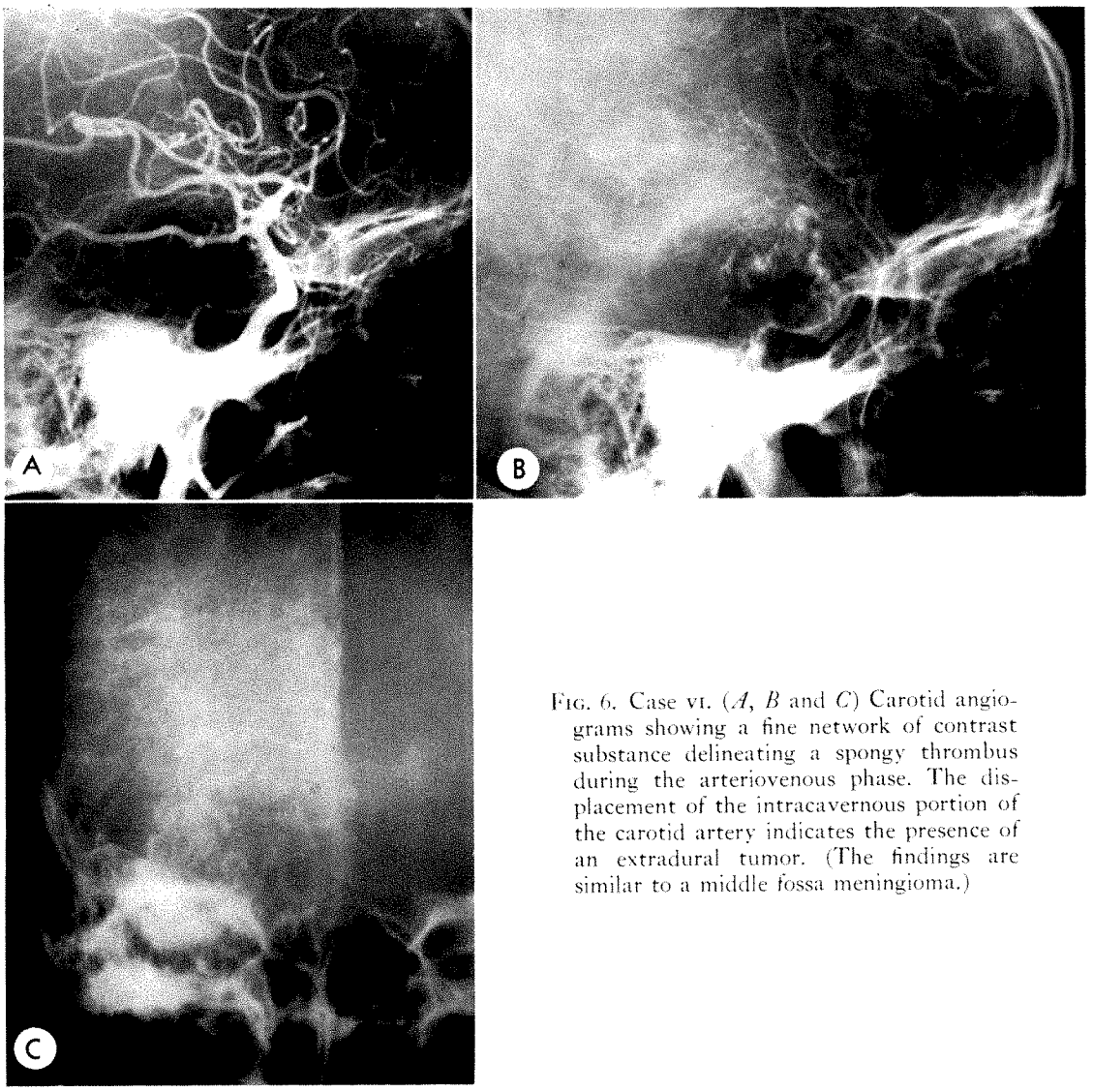


Fig. 6. Case VI. (*A*, *B* and *C*) Carotid angiograms showing a fine network of contrast substance delineating a spongy thrombus during the arteriovenous phase. The displacement of the intracavernous portion of the carotid artery indicates the presence of an extradural tumor. (The findings are similar to a middle fossa meningioma.)

tardation and "Witzelsucht." A left facial paresis was present. Papilledema and a slight increase in the tone of the right arm and leg were questionable findings. Extensor left plantar response was present.

Plain skull roentgenograms showed several curvilinear calcifications in the left frontal region associated with a series of dotted calcifications in the more cranial portions of the frontal lobe.

A left carotid angiogram showed a depressed upper loop of the siphon and a smooth backward displacement of the left anterior cerebral artery for about 5 cm. of its length, having the appearance of being stretched around a space-occupying lesion in the left frontal lobe. Superimposed upon the left anterior cerebral artery was an abnormal vessel which was wide and irregular and had upon it varices filled with contrast material in the late arterial and phlebogram phases. In particular, one vessel of this complex persisted (in the late phlebogram phase) and ran upwards and backwards to the inner table toward the bregma (Fig. 7, *A*, *B* and *C*). At operation, after removal of the right frontal lobe, a fairly large smooth plum colored tumor was seen which separated easily from the brain substance. There were several large ves-

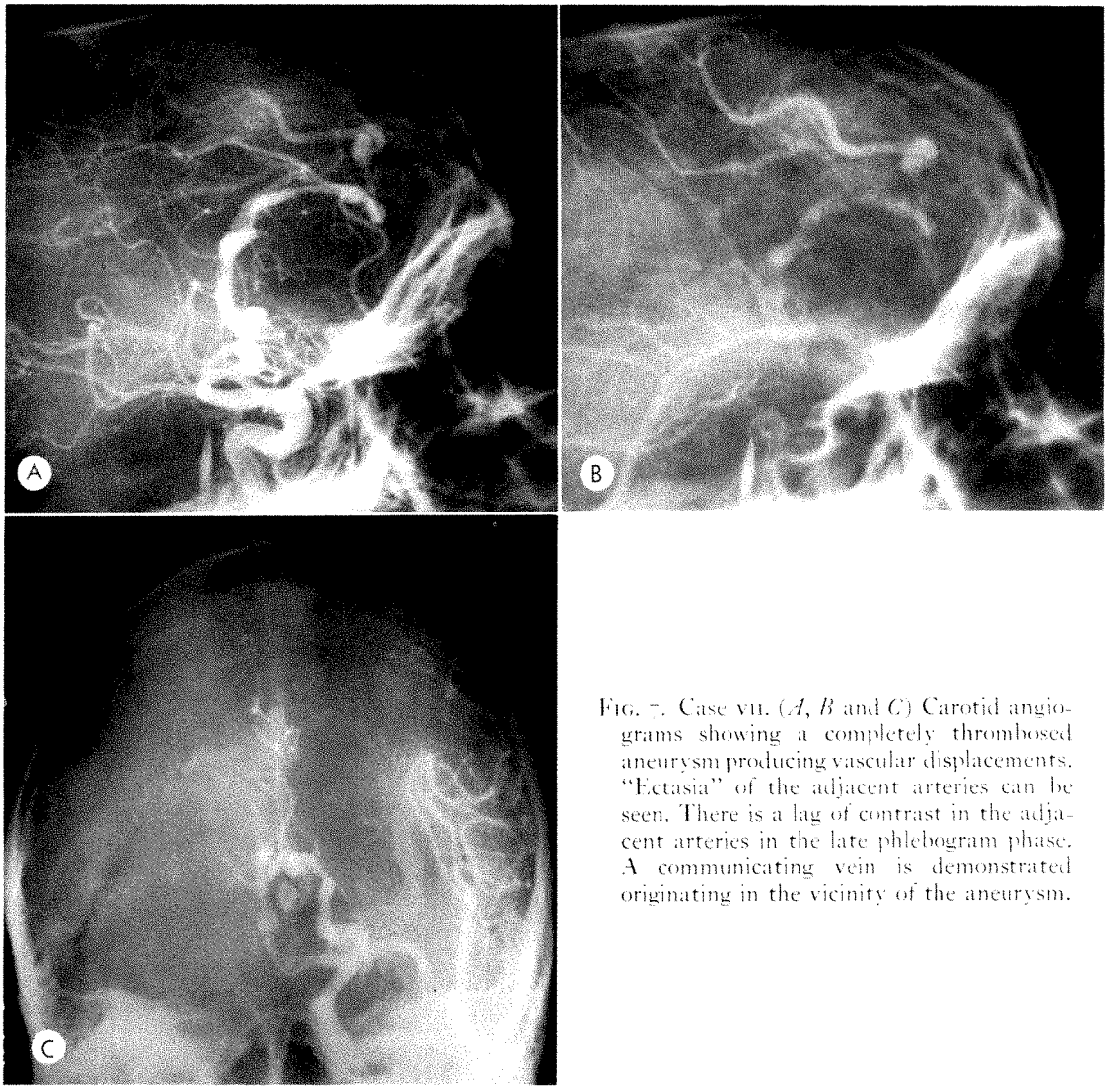


FIG. 7. Case VII. (*A*, *B* and *C*) Carotid angiograms showing a completely thrombosed aneurysm producing vascular displacements. "Ectasia" of the adjacent arteries can be seen. There is a lag of contrast in the adjacent arteries in the late phlebogram phase. A communicating vein is demonstrated originating in the vicinity of the aneurysm.

sels spread over and intimately related to the tumor. Chocolate colored blood oozed from the tumor. Large atheromatous vessels were exposed on the left which also were in close relation to the tumor. The tumor became mobile, revealing a large aneurysm.

The pathology specimen showed a large aneurysm filled with organizing thrombus. Attached to the surface of the sac were two fairly large, partly calcified arteriosclerotic arteries. In one region the two arteries were in continuity. The sac originated near the junction of the two arteries.

DISCUSSION

The correct roentgen diagnosis of aneurysmal thrombosis is difficult. Prior to cerebral angiography, aneurysms have been diagnosed in plain skull roentgenograms by their typical curvilinear calcification, which is a sequela of necrosis. Calcification may occur in any tissue layer of the aneurysm, and, if associated with changes of the inner tissue layer, thrombus formation may result. Without involvement of the intima, mural thrombosis will be absent. The satisfactory assessment of aneurysmal thrombosis by plain skull roentgenography is hardly possible since no information can be obtained about the inner wall of the aneurysm. The ready demonstration of an-

eurysms by cerebral angiography affords a more correct diagnosis.

The procedure suggested for the demonstration of a thrombus,[15] although very accurate, is complicated, time consuming and costly. The examination entails a separate injection of lipiodol into the sphenoid sinus combined with carotid angiography to demonstrate the nonthrombosed portion of an infraclinoid aneurysm.

Flecks of contrast material around an aneurysm may deserve consideration as a sign of thrombosis; however, only 1 such observation has been made since 1941.[9] In many cases contrast material around an aneurysm indicates leakage rather than contrast medium absorbed in the thrombus.

The retention of contrast material within the entire aneurysm or at the fundal portion visualized in all three phases has been explained as a cause of the decreased circulatory velocity, which is a predisposing factor for thrombus formation.[13] It has also been shown that the layering of contrast material within the aneurysm can be attributed to a lag of the opaque medium in the dependent portions of the aneurysm because of the higher specific gravity of the contrast substance.[6] Three cases in our study showed retention of contrast material in all 3 phases. A mural thrombus was present in each of these.

The most common sign in the recognition of partial thrombosis is the displacement of the arteries related to the aneurysm.[1] This is particularly true in thrombosis of cavernous sinus aneurysms in which only the patent part is demonstrated. The nonvisualized space between the displaced artery and the contrast demonstrated aneurysm represents the thrombus, which is the causative factor of vascular displacement. Curvilinear or round shaped calcification with the concavity directed towards the aneurysm is indicative of aneurysmal wall calcification. The nonvisualized portion of the aneurysm represents the thrombus. In a similar way, bone destruction or erosion occurring in the vicinity of the contrast filled aneurysm will indicate the presence of partial thrombosis. In our study it was found that the above mentioned factors may occur separately or in combination with one another.

The presence of a convex filling defect in the fundal portion of the aneurysm is likewise indicative of mural thrombosis. The thrombus may, in fact, be located in the central region of the aneurysm. A thrombus may be solid or, in an occasional case, porous. The arteriogram in such an instance shows the contrast medium in the crevices of the thrombus, simulating the appearance of pathologic vessels as seen in meningiomas. A careful evaluation of the roentgenogram will differentiate these two entities.

Complete thrombosis of an aneurysm has, in general, no roentgenographic diagnostic features[10,15] with the exception of the displacement of neighboring vessels in the presence of large aneurysms. In one of our cases of complete thrombosis, varicosity of vessels which were related to the space-consuming lesion was observed. The phlebogram phase showed evidence of venae comitantes. This particular roentgen appearance, normally associated with new growth formation, should, in the light of this case, be considered in the differential diagnosis of a thrombosed aneurysm.

SUMMARY

A series of cases of intracranial aneurysms was reviewed in an effort to establish a roentgen diagnostic feature of partial or complete aneurysmal thrombosis. It was found that in the majority of the cases displacement of vessels adjacent to the partially thrombosed aneurysm indicated quite accurately the size and location of the thrombus. Bone destruction and curvilinear calcifications in the vicinity of the aneurysm were likewise helpful in estimating the presence of a thrombus. The demonstration of semilunar and central filling defects was indicative of an aneurysm; lag of contrast material and a meningioma-like appearance in some instances signified thrombosis.

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VISUALIZATION OF VESSEL GRAFTS BY INTRAVENOUS ABDOMINAL AORTOGRAPHY*

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WHEN the description of the intravenous method of abdominal aortography and peripheral arteriography was published in 1959,[19] it was predicted that it would prove useful for evaluating the benefits, complications, and shortcomings of reconstructive vascular procedures. These expectations have been fulfilled; this report is an account of how intravenous abdominal aortography can be utilized for visualizing vascular grafts.

REPORT OF CASES

CASE I. *Thrombosis of left common iliac artery teflon graft 20 months following resection of a ruptured abdominal aortic aneurysm.* A 54 year old man, previously reported,[20] was well following resection of a large ruptured abdominal aortic aneurysm in March, 1960 (Fig. 1, *A* and *B*). In February, 1962, he began having severe intermittent claudication of the left leg. Intravenous abdominal aortography on November 20, 1962, disclosed complete occlusion of the left common iliac artery graft a short distance from the bifurcated prosthesis (Fig. 1*C*).

Exploratory operation by Dr. S. W. Moore on November 26, 1962, revealed metastatic carcinoma in the liver; the original site of this lesion could not be determined. The teflon graft contained thrombus in the left bifurcation limb and resection of the area with end-to-side anastomosis to a new teflon prosthesis resulted in excellent blood flow into the left leg. He was discharged on December 17, 1962.

CASE II. *Demonstration of intact abdominal aortic prosthesis 6 months after resection of abdominal aortic aneurysm.* A 63 year old man developed sudden severe abdominal pain 6 months following successful resection of an abdominal aortic aneurysm (Fig. 2, *A* and *B*) and replacement with a teflon graft. Intravenous abdominal aortography was performed on October 3, 1962, in order to rule out hemorrhage from the graft. It revealed an intact prosthesis (Fig. 2, *C* and *D*). Following a period of observation during which the stools were free of blood and the hematocrit normal, he was discharged.

CASE III. *Visualization of an intact homograft 6 years following resection of an abdominal aortic aneurysm.* A 65 year old man began having leg pains 6 years following resection of an abdominal aortic aneurysm (Fig. 3*A*), which had been replaced by a homograft on November 20, 1956. Intravenous abdominal aortography on June 25, 1963, (Fig. 3*B*) and aortotomography (Fig. 3*C*) showed an intact well-functioning graft.

CASE IV. *Thrombosis of left iliac limb of an aortic nylon graft 3 years following operation for occlusive disease of the abdominal aorta.* A 65 year old woman with severe obliterative arteriosclerotic disease of the abdominal aorta (Fig. 4*A*) had had a resection of the abdominal aorta with replacement by a nylon prosthetic bifurcation graft. Because of abdominal pain, intravenous abdominal aortography was performed on February 16, 1960. This revealed intact abdominal aortic blood flow except for some stenosis of the left common iliac prosthesis along the suture line to the left common iliac artery (Fig. 4*B*). Four months later, there was recurrence of the abdominal pain and an exploratory operation in June, 1960, revealed partial bowel obstruction due to adhesions. She was re-admitted in September, 1961, because of gradual onset of severe intermittent claudication of the left leg. An intravenous abdominal aortogram at this time showed complete occlusion of the left limb of the aortic prosthesis close to the bifurcation (Fig. 4*C*). This was confirmed at operation. Replacement of the nylon prosthesis with endarterectomy of the left common iliac artery beginning distal to the graft was done on October 29, 1961, and resulted in restoration of pulsations of the arteries of the left leg.

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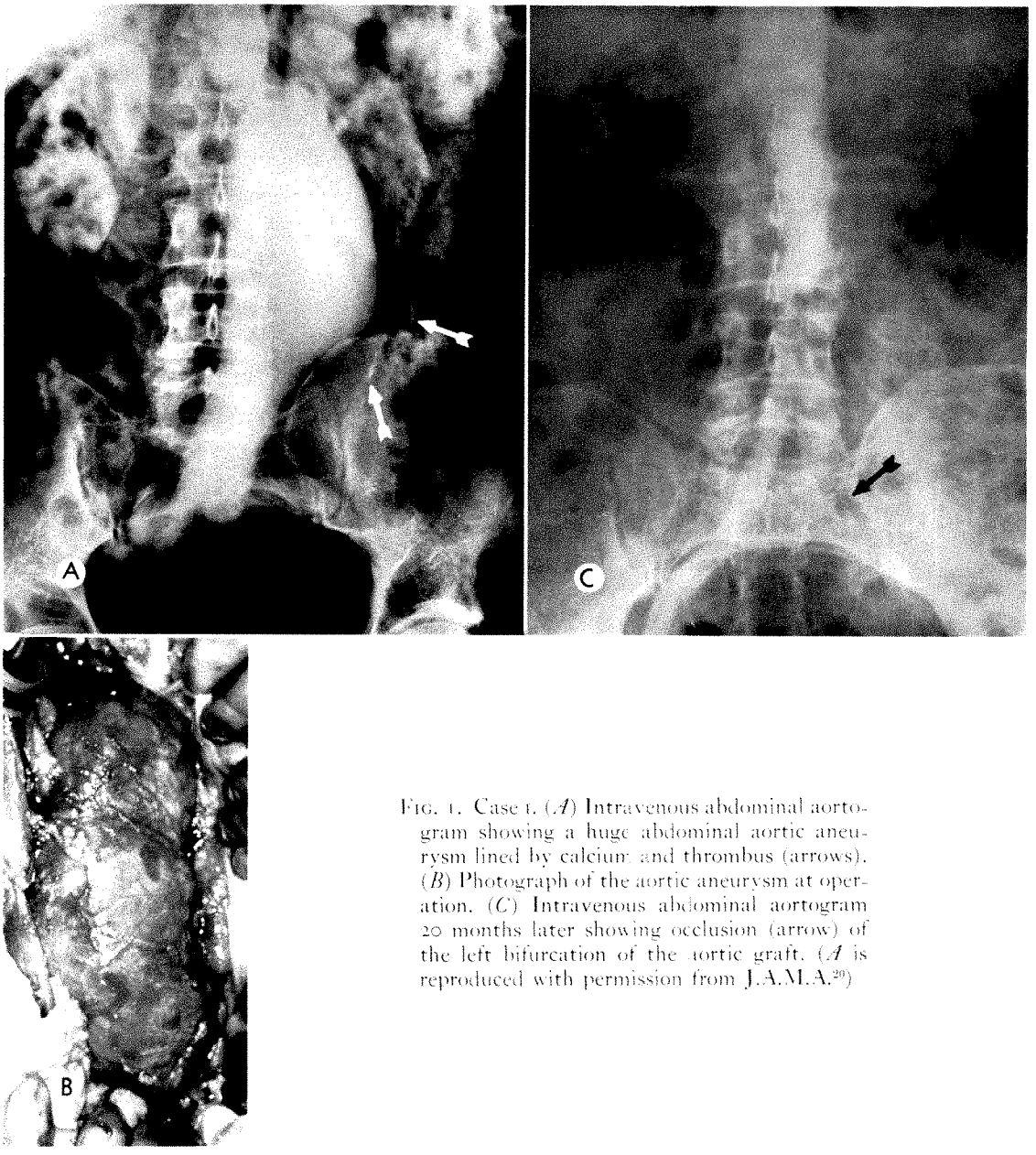


FIG. 1. Case 1. (*A*) Intravenous abdominal aortogram showing a huge abdominal aortic aneurysm lined by calcium and thrombus (arrows). (*B*) Photograph of the aortic aneurysm at operation. (*C*) Intravenous abdominal aortogram 20 months later showing occlusion (arrow) of the left bifurcation of the aortic graft. (*A* is reproduced with permission from J.A.M.A.[20])

CASE V. *Mycotic aneurysm of the abdominal aorta following Salmonella septicemia.* A 54 year old man, who had had partial resection of his stomach in 1958 for a gastric ulcer and resection of an abdominal aortic aneurysm with replacement by a teflon graft on May 24, 1962 (Fig. 5*A*), developed a mycotic aneurysm proximal to the graft. He was re-admitted on October 16, 1962, because of gastroenteritis, diarrhea, and fever. The stools were positive for Salmonella typhimurium. After treatment with chloramphenicol he became afebrile, but subsequently he had recurrent fever and the blood cultures became positive for Salmonella typhimurium. Following retreatment with antibiotic drugs he again became febrile and then developed positive blood cultures for Salmonella. On January 18, 1963, a pulsatile left upper quadrant mass was discovered. Intravenous abdominal aortography disclosed a huge saccular aneurysm of the abdominal aorta proximal to the suture line of the graft (Fig. 5*B*). Incision

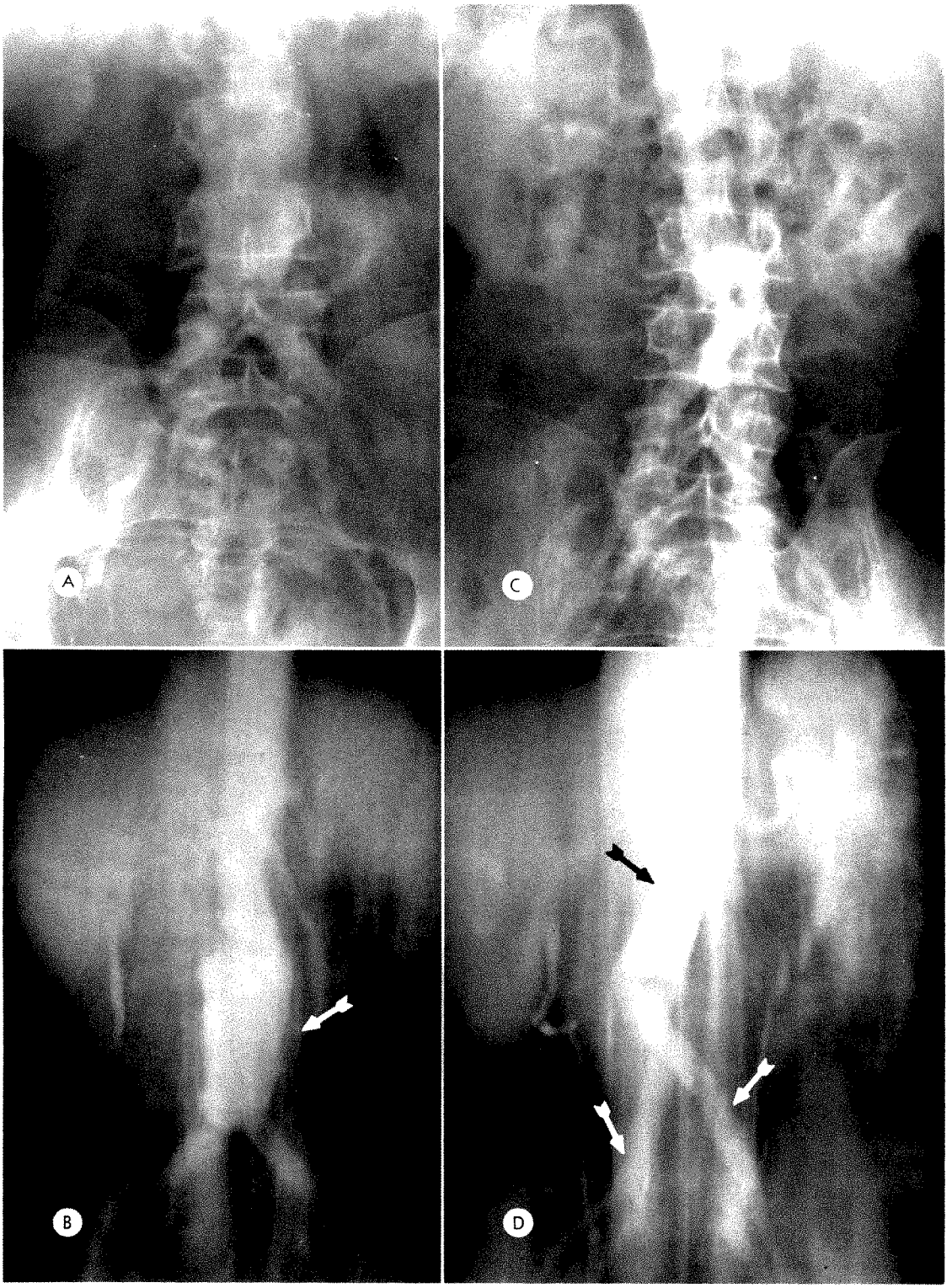


FIG. 2. Case II. (*A*) Intravenous abdominal aortogram showing an abdominal aortic aneurysm. (*B*) Intravenous aortotomogram demonstrating the large aneurysm lined by thrombus along the left wall (arrow). (*C*) Repeat intravenous abdominal aortogram, 7 months later, made because of sudden onset of abdominal pain, showing tortuosity, but without rupture of the graft. (*D*) Intravenous aortotomogram demonstrating the details of the tortuous but intact graft. Arrows point to suture lines of the graft with the aorta and iliac arteries.

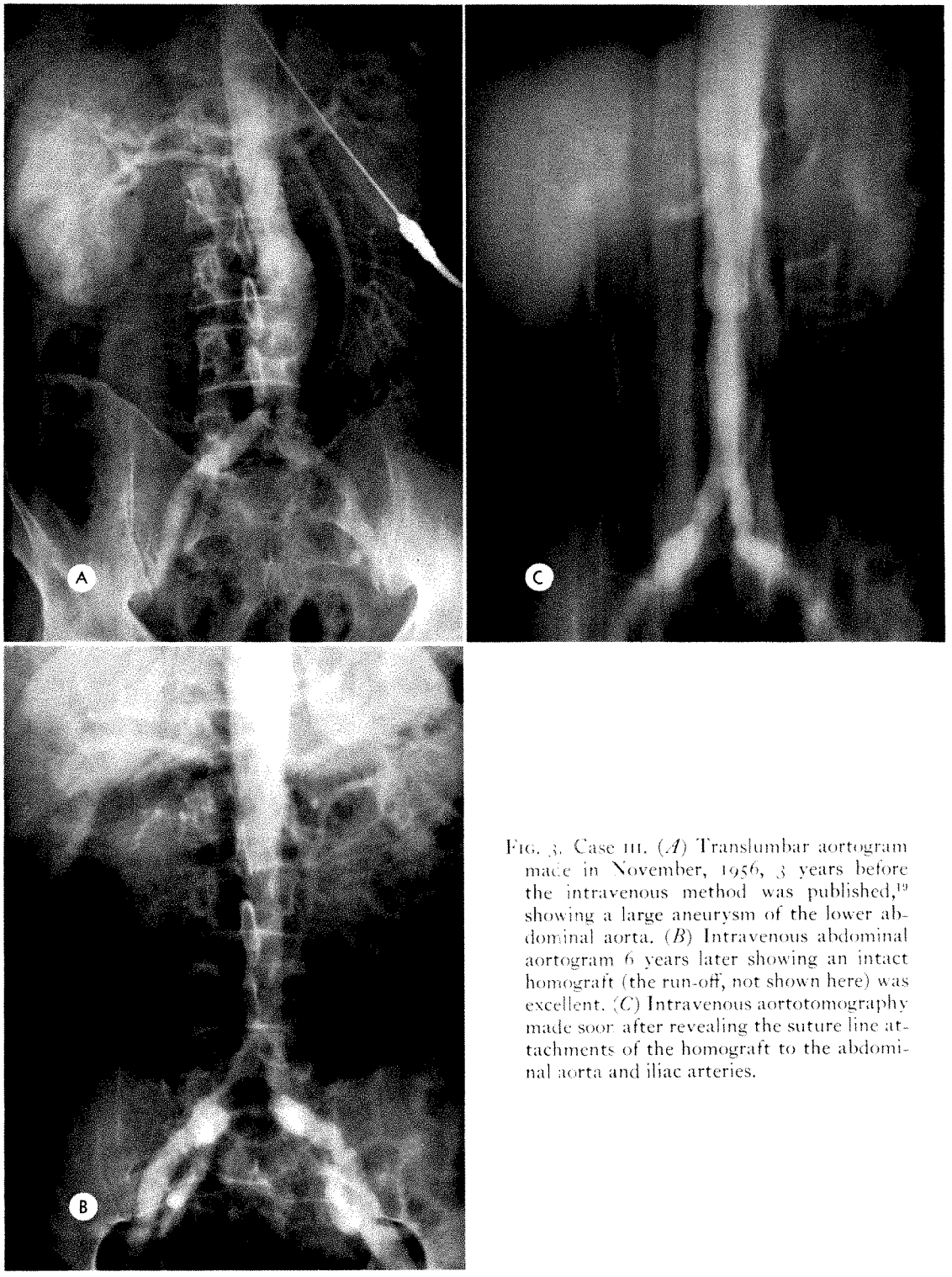


FIG. 3. Case III. (*A*) Translumbar aortogram made in November, 1956, 3 years before the intravenous method was published,[19] showing a large aneurysm of the lower abdominal aorta. (*B*) Intravenous abdominal aortogram 6 years later showing an intact homograft (the run-off, not shown here) was excellent. (*C*) Intravenous aortotomography made soon after revealing the suture line attachments of the homograft to the abdominal aorta and iliac arteries.

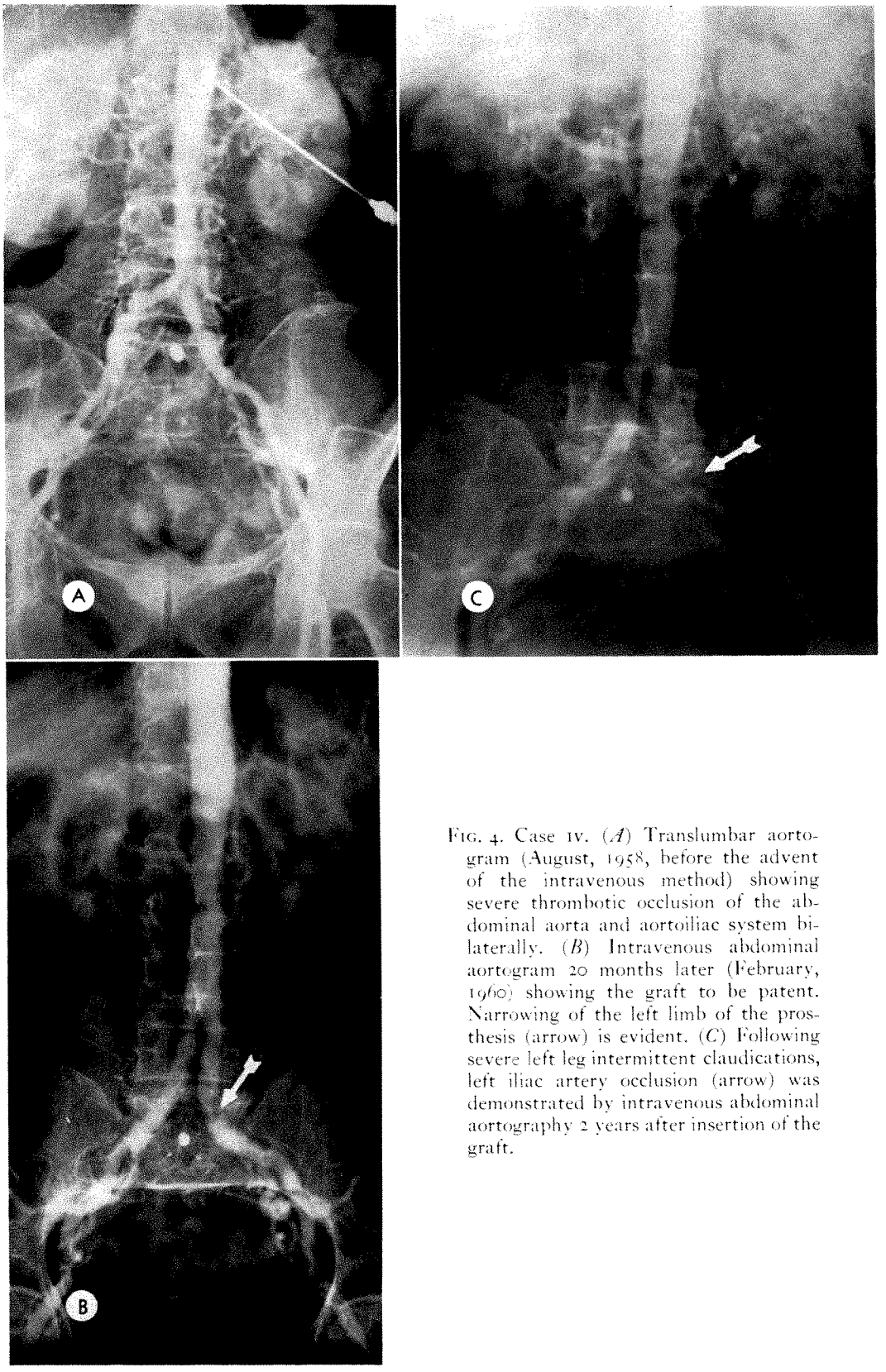


Fig. 4. Case IV. (*A*) Translumbar aortogram (August, 1958, before the advent of the intravenous method) showing severe thrombotic occlusion of the abdominal aorta and aortoiliac system bilaterally. (*B*) Intravenous abdominal aortogram 20 months later (February, 1960) showing the graft to be patent. Narrowing of the left limb of the prosthesis (arrow) is evident. (*C*) Following severe left leg intermittent claudications, left iliac artery occlusion (arrow) was demonstrated by intravenous abdominal aortography 2 years after insertion of the graft.

and drainage of several abdominal abscesses, nephrectomy of the left nonfunctioning kidney, and plication of the mycotic aneurysm of the abdominal aorta on January 19, 1963 was followed by subsidence of fever. However, he became febrile again on February 28, 1963 and, despite antibiotic therapy, died on March 5, 1963. At autopsy, there was rupture of the mycotic aneurysm.

DISCUSSION

The value of contrast serial roentgenography for assessing the results of resectional surgery for correction of thoracic aortic coarctation was recognized in this center in 1951.[5,6,11] Earlier, in 1949, contrast roentgenography had also been used to demonstrate aortic anastomoses and grafts in the experimental animal.[8,10,11,12] The wide use of angiography at the present time for demonstrating the effectiveness of vascular grafts after correctional and bypass surgery has recently been well described by DeBakey and his colleagues[1] and Sutton.[23]

Intravenous abdominal aortography, because it avoids abdominal aortic puncture necessary for translumbar aortography and retrograde catheterization of the aorta (Seldinger percutaneous femoral arterial technique), is admirably suited for visualization of the abdominal aorta.[15,21] Thereby, abdominal aortography even when there are complications of the grafts such as aneurysms or thrombosis can be accomplished. Javid *et al.*[9] and others[2,3,7,16,18] have recently reported such complications.

Aneurysm and rupture of an abdominal aortic graft visualized by intravenous aortography are shown in Figure 5*B*. Accompanying aortograms show thromboses of prosthesis following resection of an aortic abdominal aneurysm (Fig. 1*C*, Case 1), occlusive disease of the abdominal aorta

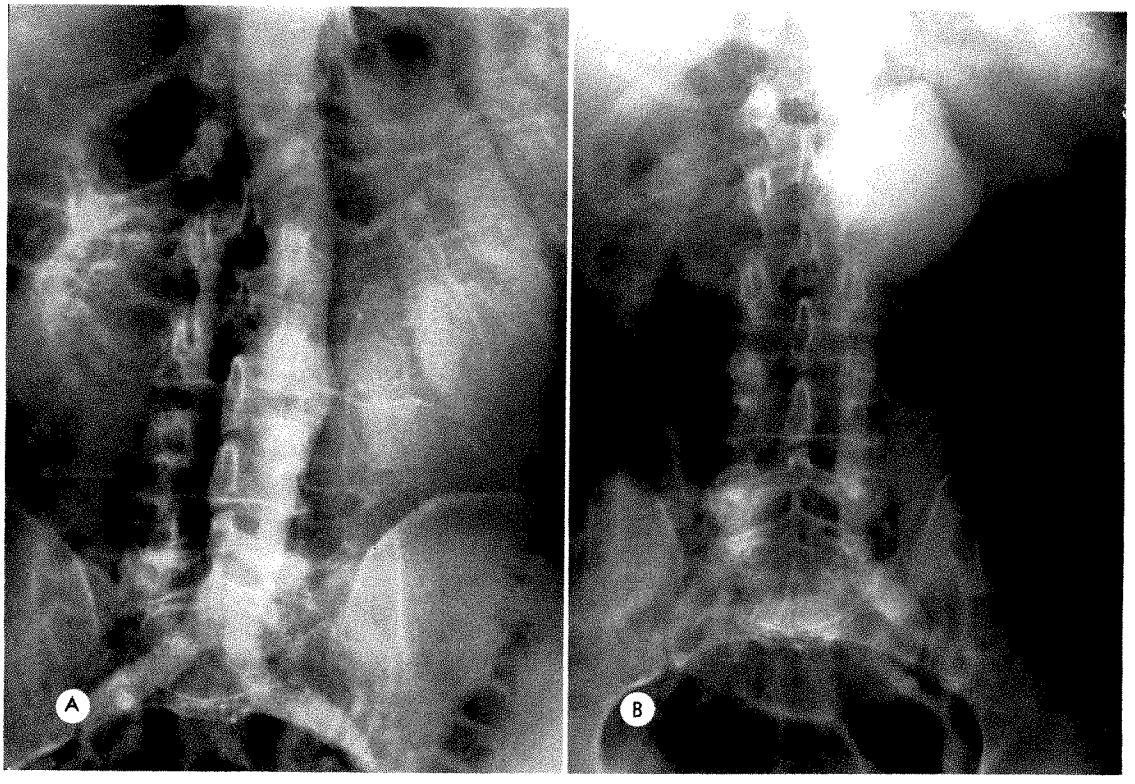


Fig. 5. Case v. (*A*) Intravenous abdominal aortogram showing an abdominal aortic aneurysm. (*B*) Intravenous abdominal aortogram 9 months later revealing a huge mycotic aneurysm with thrombus above the suture line of the proximal portion of the teflon graft. Note the absence of the left renal artery and nonopacification of the left kidney.

(Fig. 4*C*, Case IV) and an intact aortic homograft (Fig. 3*B*, Case III). Exquisite details of the graft including the suture lines were seen after aortotomography (Fig. 3*C*). The technique of intravenous aortotomography was recently published.[22] Two patients with abdominal pain were suspected of having rupture of the grafts (Fig. 2, *C* and *D*, Case II; and 4*B*, Case IV), but intravenous aortography revealed intact prostheses. In Case IV, however, stenosis at the terminal portion of the graft was demonstrated (Fig. 4*B*), and this subsequently was the site of an extensive occlusive lesion (Fig. 4*C*).

SUMMARY AND CONCLUSIONS

Because it avoids aortic or arterial puncture, intravenous abdominal aortography is useful and probably safer than translumbar and retrograde aortography for visualization of abdominal aortic grafts following resectional surgery of aneurysms and thrombotic occlusive disease of the abdominal aorta. The value of intravenous abdominal aortography, especially intravenous aortotomography, is well illustrated in 4 patients. In 3 individuals, abdominal aortic grafts were inserted after resection of abdominal aortic aneurysm. In the other patient, the graft was made following resection of thrombotic occlusive disease of the abdominal aorta. In one patient, with an intact homograft which has functioned for 6 years, exquisite detail of the prosthesis and the suture lines was demonstrated by intravenous aortotomography. Two patients presenting with abdominal pain after insertions of aortic grafts following aortic resections for aneurysm and occlusive disease, respectively, were found to have fairly intact prostheses. In the latter case, however, partial stenosis of the left limb of the prosthesis was subsequently (a year later) the site of complete occlusion. Intravenous abdominal aortography proved to be the ideal method for demonstrating a mycotic abdominal aneurysm due to Salmonella typhimurium which developed in a teflon graft that had replaced an arteriosclerotic aneurysm.

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EVALUATION OF THE DEGREE OF AORTIC VALVE STENOSIS BY DIRECT ROENTGENOLOGIC MEASUREMENT OF THE OSTIUM*

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RETROGRADE catheterization of the thoracic aorta had its origin in the work of Radner[56] and was given great impetus by the technique of Seldinger[60] which permitted easy percutaneous catheterization of peripheral arteries. Aortography at first was used to evaluate coarctation of the aorta or patent ductus arteriosus[17,39] and later to diagnose aortic insufficiency[19] and estimate its severity.[44] Following initial roentgenographic observations regarding gross changes in the thoracic aorta in patients with aortic valve stenosis,[18,25,32] Castenfors *et al.*,[21] Ödman and Philipson,[54] and Kjellberg *et al.*[42] detailed the morphologic and hemodynamic abnormalities in the valve itself demonstrable angiographically in patients with this disease. Ödman and Philipson described the radiolucent jet of unopacified blood ejected into the opacified aorta through the stenosed valve leaflets and stated "it ought to be possible to estimate the width of the orifice on the basis of the jet's calibre."

The purpose of this paper is to present our findings in 22 patients with aortic valve stenosis in whom a jet could be measured during thoracic aortography (Fig. 1). The size of the jet at its base, corresponding to the size of the valve ostium, was corrected for roentgenographic magnification, calculated per square meter of body surface area of the patient, and correlated with other clinical, roentgenologic, hemodynamic, and surgical data. A program for the preoperative evaluation of patients with suspected aortic valve stenosis has evolved.

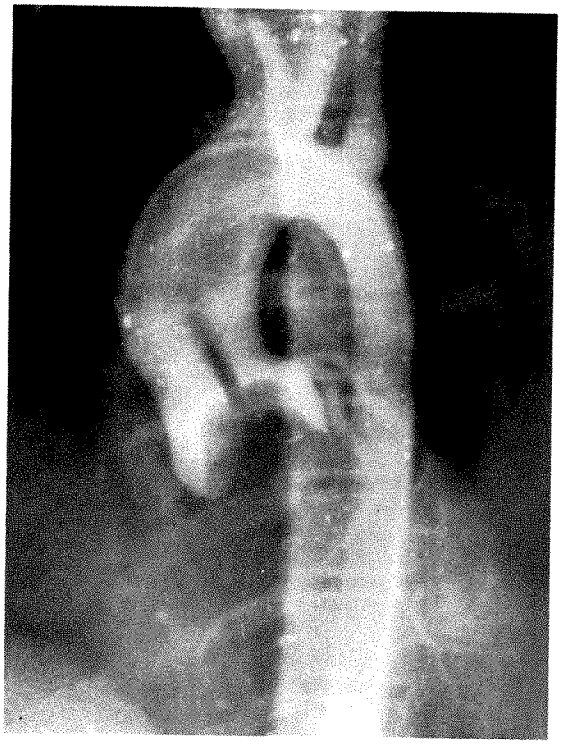


FIG. 1. Case 2. Thoracic aortogram in a 12 year old girl with congenital aortic valve stenosis. Patient in slight right posterior oblique position. The examination shows doming of aortic valve leaflets and a radiolucent jet produced by ejection of unopacified blood from the left ventricle through the stenosed valve leaflets into the opacified aorta. The area of the valve ostium is 3 per cent of the area of the aorta. There is waist formation at the base of the aorta and poststenotic dilatation of the more distal ascending aorta.

MATERIALS AND METHODS

Included in this study were 22 patients with aortic valve stenosis, 16 males and 6 females. Patients ranged in age from 2 to 52

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years, with only 2 being more than 19 years old. Congenital stenosis was present in 19 patients and acquired rheumatic stenosis in 3 (Cases 3, 6 and 7). The severity of stenosis was rated clinically on the basis of 0 to 4 plus, with particular emphasis placed on dyspnea, exercise tolerance, syncope, precordial pain, congestive heart failure, and electrocardiographic findings. In all but 1 patient conventional roentgenograms of the chest were made in posteroanterior, left lateral, and right and left anterior oblique positions following barium swallow. In these patients cardiac size, left ventricular hypertrophy, poststenotic dilatation of the ascending aorta, left atrial size and evidence in the lungs of left heart failure were rated on the basis of 0–4 plus.

All patients had thoracic aortography for evaluation of the aortic valve, and 10 had preceding right heart catheterization and pulmonary artery angiocardiography. Examinations were done in children under general anesthesia and in patients over 10 years old under local lidocaine hydrochloride[a] anesthesia with diphenhydramine hydrochloride[b] and meperidine hydrochloride[c] premedication. All studies were performed by cut-down or percutaneous insertion of #6F-10F NIH or #9F radiopaque Teflon catheters[d] or radiopaque yellow Ödman-Ledin tubing[e] in a common femoral artery or vein or antecubital vein. During catheterization pressure recordings were obtained with a Statham physiologic pressure transducer,[f] and oxygen saturations were obtained with a Model XC-50B oximeter cuvette.[g] In those patients in whom the catheter could be passed through the stenosed aortic valve, a pressure gradient between the aorta and left ventricle was obtained prior to angiography. The catheter was then positioned 3 to 5 cm. above the valve for thoracic aortography.

For pulmonary artery angiocardiography 1.2–1.5 ml. of 75 per cent sodium diatrizoate[h] and for aortography 0.8–1.0 ml. of 75 per cent sodium diatrizoate or 60 per cent methylglucamine diatrizoate[i] per kilogram of body weight were injected under 4–9 kg. per sq. cm. pressure, using a Gidlund automatic injector, in order to deliver 20–25 ml. of contrast material per second. Simultaneous biplane roentgenography was done with the Schönander cut film changer at a rate of 4–6 pairs of films per second for pulmonary artery angiocardiography and 6 pairs of films per second for thoracic aortography. For pulmonary artery angiocardiography all patients were supine, and roentgenograms were made in the anteroposterior and left lateral projections. For thoracic aortography 16 patients were supine, and roentgenograms were made in the anteroposterior and left lateral projections; the remaining 6 patients were placed in an approximately 30 degree right posterior oblique position, and right posterior oblique and left posterior oblique roentgenograms were obtained. Duration of injection of the contrast material and times of film exposures were recorded on a continuous electrocardiographic tracing during the procedures.

In all patients having pulmonary artery angiocardiography, the left atrial volumes were calculated during maximum atrial diastole by the method of Arvidsson and Ödman,[4] and thickness of the left ventricle in the mid-portion of its left lateral border was measured during maximum ventricular diastole. The cross-sectional diameter of the aortic jet was measured in the projection in which its appearance was most distinct. Of the 16 patients who were in the supine position, the jet was measured in the anteroposterior projection in 14 and in the lateral projection in 2. Of the 6 patients who were

[a] Xylocaine hydrochloride, Astra Pharmaceutical Products, Incorporated, Worcester, Massachusetts.
[b] Benadryl, Parke Davis and Company, Detroit, Michigan.
[c] Demerol, Breon Laboratories, New York, New York.
[d] Supplied by United States Catheter and Instrument Corporation, Glens Falls, New York.
[e] Supplied by KIFA Instrument and Equipment Company of Sweden through Schick X-ray Company, Incorporated, 205 West Wacker Drive, Chicago, Illinois.
[f] Statham Transducers, Inc., Hato Rey, Puerto Rico.
[g] The Waters Corporation, Rochester, Minnesota.
[h] Hypaque, Winthrop Laboratories, 1450 Broadway, New York 18, New York.
[i] Renografin, E. R. Squibb and Sons, 745 Fifth Avenue, New York 22, New York.

in the right posterior oblique position, the jet was measured in this projection in 5 and in the left posterior oblique projection in 1.

No allowance was made for the contribution to the apparent diameter of the jet made by the thickness of the radiopaque aortic valve leaflets in the open position. The cross-sectional diameters and areas of the aorta immediately above the sinuses of Valsalva and at the level of maximum poststenotic dilatation were measured during ventricular systole, using right angle projections. All measurements were corrected for roentgenographic magnification and expressed per square meter of body surface area.

RESULTS (TABLE I)

Thirteen of the 22 patients were entirely asymptomatic and, except for systolic ejection murmurs, had no clinical evidence of aortic stenosis. Of the remaining 9 patients, the clinical severity of the heart disease was judged to be one plus in 5, two plus in 3, and four plus in 1 patient. Four of the 9 symptomatic patients had additional heart valve lesions. Clinical symptoms and electrocardiographic abnormalities tended to occur in patients in whom the area of the valve ostium was less than 10 and 15 per cent, respectively, of the area of the aorta immediately above the valve, but no strict correlations were demonstrated. In conventional roentgenograms of the chest, there was no correlation between the severity of stenosis and the degree of left ventricular hypertrophy, poststenotic dilatation of the ascending aorta, left atrial enlargement or pulmonary venous congestion. The cardiothoracic ratio in postero-anterior roentgenograms of the chest was less than 50 per cent in all patients with uncomplicated aortic valve stenosis and was greater than 50 per cent only in patients with additional valve lesions.

Angiographically, the diameter of the aortic jet at its base was less than 12 mm. per square meter of body surface area in all patients in this series. The area of the ostium, calculated from the diameter of the jet and assuming the jet was round, ranged from 7.2 to 84 sq. mm. per square meter of body surface area. In 20 of 22 patients the area of the ostium was less than 60 sq. mm. per square meter of body surface area. The cross-sectional area of the aorta immediately above the valve was 101–630 sq. mm. per square meter of body surface area. The aortic area in 19 of 22 patients was between 200 and 470 sq. mm. This is in agreement with calculations made in control patients without aortic valve disease in whom the maximum aortic area immediately above the valve was 300 ± 150 sq. mm. per square meter of body surface area. The cross-sectional area of the ascending aorta at the level of maximum poststenotic dilatation was 296–1,050 sq. mm. per square meter of body surface area. In 20 of 22 patients the area of poststenotic dilatation was greater than 450 sq. mm. per square meter of body surface area. Thickness of the left ventricular wall measured during maximum ventricular diastole was 5.3–9.3 mm. per square meter of body surface area. In 10 of 11 patients the left atrial volume was 25–49 ml. per square meter of body surface area. Left atrial volumes, therefore, were at the upper limits of normal or slightly enlarged, since the normal left atrial volume calculated in control patients was 30 ± 10 ml. per square meter of body surface area. This confirms the findings of others[35,40,45] that the left atrium may be slightly enlarged in some patients with aortic valve stenosis. There was no correlation between the area of the valve ostium and the thickness of the left ventricular muscle, the volume of the left atrium, or the degree of poststenotic dilatation of the aorta.

The diameter of the aortic jet at its base, corresponding to the valve ostium, was 14–62 per cent of the diameter of the aorta measured immediately above the valve. Twenty of 22 patients had diameters less than 50 per cent. More important, the area of the valve ostium was 2–35 per cent of the area of the aorta, and 20 of 22 patients had areas less than one-fourth of the aortic area. The angiographic findings correlated quite

TABLE I

EVALUATION OF THE DEGREE OF AORTIC VALVE STENOSIS BY DIRECT ROENTGENOLOGIC MEASUREMENT OF THE OSTIUM

Patient	Age	Sex	BSA	Clinical Severity	ECG	Conventional Chest Roentgenograms					Jet Diameter before Corrections (mm.)	Jet Diameter (mm./m.²)	Jet Area (sq. mm./m.²)	Ao Diameter (mm./m.²)	Ao Area (sq. mm./m.²)	PS Dilat. (sq. mm./m.²)	LV (mm./m.²)	LA (ml./m.²)	Diameter of Jet	Area of Jet	Gradient	Surgery	Comment
						C–T Ratio	LVH	PS Dilat.	LA Size	Pulm. V.									Diameter of Ao (per cent)‡	Area of Ao (per cent)‡			
1 BC	16	M	1.5	1+	?LVE	42	2+	2+	N	N	5	2.5	7.2	18.1	390	563	—	—	14	2	—	—	—
2 BH	12	F	1.14	1+	LVE	46	2+	0	3+	N	5	3.3	9.7	20.3	314	487	8.8	25	16	3	—	0.5 cm.	—
3 LN	45	M	1.9	2+	LVE	53	1+	2+	N	N	9	3.5	18	17.5	430	455	—	—	20	4	—	—	AoI, MI
4 DF	11	F	1.2	0	N	48	3+	2+	N	N	7	4.1	15.7	18.7	319	595	5.8	30	22	5	—	0.6 cm.	—
5 SR	11	M	1.2	2+	?LVE	44	1+	3+	N	N	7	4.3	17.7	19.9	305	517	—	—	22	5	—	tight	—
6 EB	52	F	1.47	4+	—*	50	1+	3+	3+	4+	10.5	4.9	27.8	18.9	440	630	—	—	26	6	—	—	AoI, MS, MI
7 GG	10	M	1.15	0	N	45	3+	4+	1+	N	9	6.1	33.4	23.6	517	1030	5.4	32	26	7	—	—	—
8 PT	9	F	0.87	0	?LVE	47	3+	2+	1+	N	8	7.1	34.4	26.7	470	734	9.0	29	27	7	—	0.6 cm.	—
9 DC	18	F	1.4	1+	LVE	55	3+	2+	3+	1+	10	4.4	23	16.1	304	567	—	—	28	8	—	—	AoI
10 CB	8	M	1.06	0	N	47	3+	2+	N	N	8	5.8	26.8	20	311	547	7.3	35	29	9	—	~1 cm.	—
11 KW	4.5	M	0.74	2+	N	50	2+	2+	1+	1+	7	7.6	30.7	26	332	685	7.4	41	29	9	35	~1 cm.	—
12 JM	7	M	0.93	1+	LVE	58	3+	4+	1+	1+	10.5	8.7	56	29.1	630	1050	7.0	37	30	9	27	—	AoI (cong.)
13 TB	10	M	1.18	0	?LVE	46	3+	3+	1+	1+	10	6.3	36.4	20.4	380	660	6.1	38	30	10	58	~1 cm.	—
14 AM	2	M	0.58	0	N	—	—	—	—	—	8	11	55.5	34.6	432	583	9.3	17.5	32	13	—	—	—
15 TH	9	M	0.91	0	?LVE	47	3+	1+	2+	N	11	9	59	26.2	430	560	7.3	—	34	13	54	~1 cm.	—
16 GT	10	F	1.22	0	N	40	2+	3+	—	3+	10	5.7	31.4	14.7	212	477	6.8	—	39	15	—	~1 cm.	—
17 SJ	7	M	0.86	0	N	45	2+	2+	1+	1+	9.5	8.6	50	21.6	344	463	—	—	40	15	29	—	—
18 JH	12	M	1.32	0	—†	49	4+	0	1+	1+	11.5	6.7	46	16.9	270	473	7	46	40	17	25	—	—
19 DM	3.5	M	0.47	0	N	50	3+	0	2+	N	7	11.7	51.5	26.8	253	—	—	—	44	20	—	—	—
20 RP	14.5	M	1.82	1+	?LVE	47	3+	3+	1+	1+	19	7.7	84	16.9	304	508	5.3	—	45	21	45	—	—
21 AM	8.5	M	0.90	0	N	—	3+	3+	2+	N	13	10.2	77	17.7	212	318	7.5	49	58	35	—	—	—
22 PK	19	M	2.08	0	N	42	1+	0	N	N	13	4.4	35.6	7.1	101	296	—	—	62	35	—	—	—

* Atrial fibrillation—consistent with anterolateral infarct. Left ventricular strain pattern.
† Wolff-Parkinson-White.
‡ Percentage rounded to nearest whole number.
BSA, body surface area; ECG, electrocardiogram; LVE, left ventricular enlargement; C–T Ratio, cardio-thoracic ratio; LVH, left ventricular hypertrophy; PS Dilat., poststenotic dilatation of aorta; LA size, left atrial size; Pulm. V., size of pulmonary veins; Ao, aortic; LV, thickness of left ventricular wall; LA, left atrial volumn; Gradient, systolic pressure gradient between left ventricle and ascending aorta at cardiac catheterization; Surgery, estimation of area of valve ostium by surgeon at corrective surgery; AoI, aortic insufficiency; MI mitra insufficiency; MS, mitral stenosis, ~ indicates "approximately or slightly greater than"; cong., congenital.

well with the degree of stenosis found during surgery in the 9 patients in whom an estimate of the size of the valve opening was mentioned in operative reports.

In all patients the diameter of the poststenotic dilatation of the aorta in right angle views was identical in each projection (Fig. 2, *A–D*). This tends to confirm the belief that the poststenotic dilatation is primarily the result of unopposed lateral centrifugal forces which are exerted by opposing, counter-rotating, more or less circular backflow currents, rather than the result of the jet force directly striking the aortic wall.[62]

CLINICAL EVALUATION OF THE PATIENT

The diagnosis of congenital or acquired aortic valve stenosis usually can be made with a high degree of accuracy by clinical examination of the patient and conventional roentgenography of the chest. However, it is impossible to determine accurately the hemodynamic significance of the abnormality in these patients by ordinary clinical methods. There is no reliable correlation between the severity of stenosis and the patient's clinical status, electrocardiogram, or conventional roentgenograms of the chest. Significant aortic stenosis frequently is found in entirely asymptomatic patients.[47,63] Although electrocardiographic changes of left ventricular hypertrophy[63] or T wave negativity over the left ventricle[9] may correlate to some extent with the systolic pressure gradient, patients may have severe aortic stenosis with normal electrocardiograms[63] or be in critical condition with minimal electrocardiographic abnormalities.[57] In adults the severity of stenosis, as indicated by the transvalvar systolic pressure gradient, does not correlate with either the degree of cardiac enlargement,[40,45] left ventricular hypertrophy,[45] or poststenotic dilatation of the aorta[40,45,62] on conventional roentgenograms of the chest. Neither does the aortic valve area, estimated at cardiac catheterization, appear to correlate with these roentgen findings.[45] Furthermore, the presence of poststenotic dilatation cannot always be used as a differential feature distinguishing valvar from subvalvar aortic stenosis.[40]

PRESSURE GRADIENT ACROSS THE VALVE

In order to select patients properly for surgical correction of aortic valve stenosis, it is necessary preoperatively to determine its severity. This cannot be done by clinical evaluation of the patient or by evaluation of the aortic pulse pressure.[36] The systolic pressure gradient across the valve is considered the most reliable criterion indicating the hemodynamic significance of the stenosis. A gradient of 50 mm. Hg or more is considered adequate indication for corrective surgery. However, in patients with aortic valve stenosis, none of the techniques currently available for obtaining a pressure recording in the left ventricle is entirely satisfactory. Left ventricular catheterization via percutaneous paravertebral transthoracic puncture of the left atrium[7,26] has a high morbidity and not insignificant mortality.[5,6,12,36,51] Transmitral catheterization of the left ventricle is possible after transseptal left atrial puncture.[14,59] However, in patients with isolated aortic valve stenosis, puncture of the relatively small left atrium may be difficult, and the catheter often does not remain in the left ventricle in patients with associated mitral insufficiency. Pressure records may be damped when a small catheter is used,[11] and increasing the size of the catheter probably increases the risk of the procedure. Furthermore, it is not always possible to pass the catheter into the aorta for a pressure recording. Direct percutaneous puncture of the left ventricle[13,46] has a relatively high risk,[16,27,36] and retrograde catheterization through the aortic valve[24,66] is not always successful in patients with aortic stenosis.[2,30,33,36,48,63]

Furthermore, the systolic pressure gradient between the left ventricle and the aorta or a peripheral artery is inadequate in itself for complete evaluation of a patient with suspected aortic stenosis. A pull-back pressure recording is required to distinguish

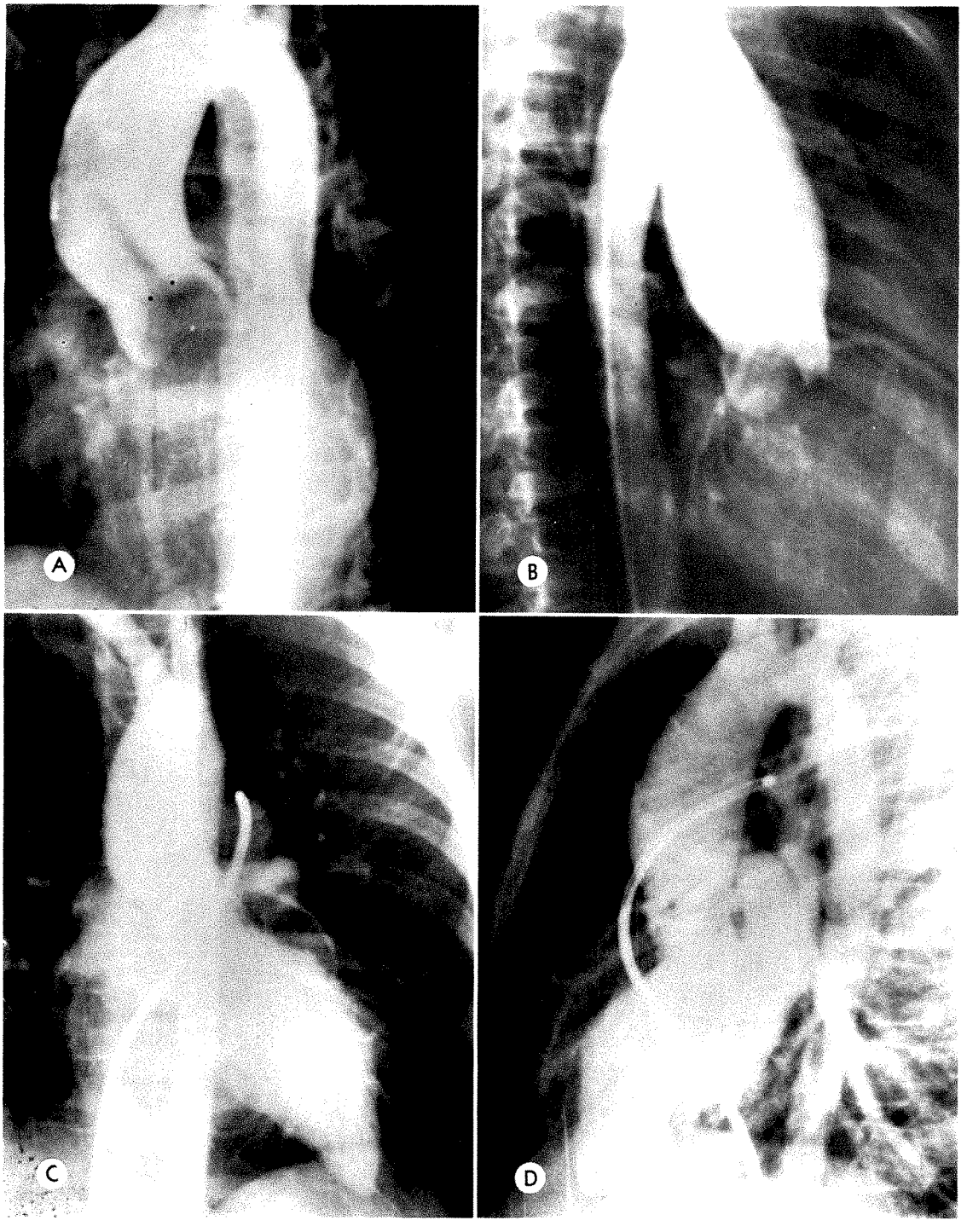


FIG. 2. Case 4. (*A*) Slight right posterior oblique and (*B*) slight left posterior oblique thoracic aortograms in an 11 year old girl with congenital aortic valve stenosis. The area of the valve ostium, as calculated from the diameter of the jet, is 5 per cent of the area of the aorta. Doming of aortic valve leaflets and poststenotic dilatation of the ascending aorta are demonstrated. The diameter of the poststenotic dilatation is identical in right angle views, indicating that it is primarily the result of unopposed lateral centrifugal forces exerted upon the aortic wall.[62] (*C*) Anteroposterior and (*D*) left lateral pulmonary artery angiocardiograms with the patient supine. Right heart catheterization and pulmonary artery angiocardiography are performed to exclude subvalvar and supravalvar aortic stenosis and other cardiac abnormalities.

subvalvar, valvar and supravalvar aortic stenosis. In addition, aortic[9,22,54] and transvalvar left ventricular pressures are subject to error. The left ventricular pressure may be misleadingly low or normal in patients who have associated abnormalities which decrease the cardiac output, such as mitral stenosis or left ventricular failure. Systolic pressure in the left ventricle may be exaggerated, if it is obtained via a retrograde catheter directed through a tight aortic valve stenosis.[9] The conditions under which the pressures are obtained, such as the stress of the examination or general anesthesia, may introduce hemodynamic artifacts.[36] Also, the wide variations in responses of individuals to similar pathologic processes may be responsible for lack of correlation of the pressure gradient with the severity of the stenosis.

Although the size of the stenotic orifice may be estimated, using the Gorlin formula,[28] from the systolic pressure gradient between the left ventricle and aorta with a simultaneous cardiac output determination, these measurements are difficult to obtain and subject to many variables and possible errors, as indicated.

ANGIOCARDIOGRAPHY

An angiocardiographic technique complementary to cardiac catheterization, therefore, is desirable for the preoperative evaluation of patients suspected clinically of having aortic valve stenosis. The techniques currently utilized are: (1) venous angiocardiography,[20,23,32,58] (2) pulmonary artery angiocardiography,[4,38,41] (3) transseptal catheterization of the left heart at the atrial level,[14,15,59] (4) percutaneous transthoracic puncture of the left atrium[5,10] or the left ventricle,[3,9,43,46,52,53,61] (5) retrograde catheterization of the left ventricle through the aortic valve,[2,24,33,36,48,63,64] and (6) retrograde thoracic aortography.

All techniques which require injection of contrast material at some point proximal to the aortic valve result in relatively poor definition of the valve ostium and make it impossible to demonstrate the aortic jet in more than one roentgenogram, if at all. Venous angiocardiography may show some of the more gross changes in the left ventricle and ascending aorta, and may localize the site of obstruction by exclusion. However, details of the valvular apparatus cannot be directly observed, and information regarding the severity of the disease is not provided. Furthermore, it is not possible with this technique to survey the heart in detail for additional abnormalities. Although pulmonary artery angiocardiography may evaluate the aortic valve in some detail, including demonstration of thickened aortic valve leaflets and doming of the funnel-shaped cusps into the aorta,[41] the size of the ostium cannot be defined clearly (Fig. 3, *A* and *B*).

Transseptal left heart catheterization is not without risk,[1,55] and its place in the pediatric age group has not yet been established. Percutaneous transthoracic puncture of the left atrium or left ventricle with angiocardiography may demonstrate some morphologic abnormalities of the aortic valve, such as thickened leaflets,[46] but the morbidity and mortality of this procedure are relatively high.[31] This is especially true when a needle large enough for angiocardiography is used[5,8] or when catheterization of the aorta for pressure recordings is done via the left ventricular puncture.[27] Also, with this method only a limited survey is possible of other cardiac abnormalities. Retrograde transvalvar catheterization of the left ventricle frequently is not possible in the presence of aortic stenosis,[2,30,33,36,48,63] and, furthermore, detailed evaluation of the valve is difficult with a catheter positioned through it.

On the other hand, in patients with aortic valve stenosis, thoracic aortography permits accurate roentgenographic assessment of the morphologic and hemodynamic abnormalities involving the valve apparatus and ascending aorta. The morphology and function of the valve leaflets can be studied during the various phases of numerous cardiac cycles. The abnormalities we have noted are identical to those described in detail by Ödman and Philipson,[54] and in many respects are similar to those de-

scribed in patients with congenital pulmonary valve stenosis.[37] In aortic valve stenosis, the amplitude and speed of movement of the valve leaflets may be decreased. In some patients with rheumatic aortic stenosis, the leaflets may appear entirely stationary. When the leaflets retract and become contiguous during ventricular diastole, they may appear unusually thick. During ventricular systole they balloon into the aorta, and a radiolucent jet may be demonstrated due to ejection of unopacified blood from the left ventricle through the stenosed ostium into the opacified aorta (Fig. 1–4, inclusive). There are wide individual variations in the caliber, distinction, direction, and duration of the jet, as well as in its position in the valve orifice. The jet is demonstrated more frequently and with greater distinction in patients with the pliable leaflets of congenital stenosis than with the more rigid, immobile leaflets of acquired rheumatic stenosis. The latter patients usually are larger in size and have relatively greater left ventricular decompensation, factors which contribute to less distinct visualization of the jet. Left coronary artery predominance, associated with left ventricular hypertrophy, usually is demonstrated. Hemodynamically, whirls and eddies of unopacified blood from the left ventricle may be demonstrated in the ascending aorta (Fig. 4) with delayed washout of contrast material from the aorta after the end of the injection. There is poststenotic dilatation of the ascending aorta, especially in its middle segment (Fig. 1–5, inclusive), and in some patients unusually active pulsations of this segment. At times slight narrowing (waist forma-

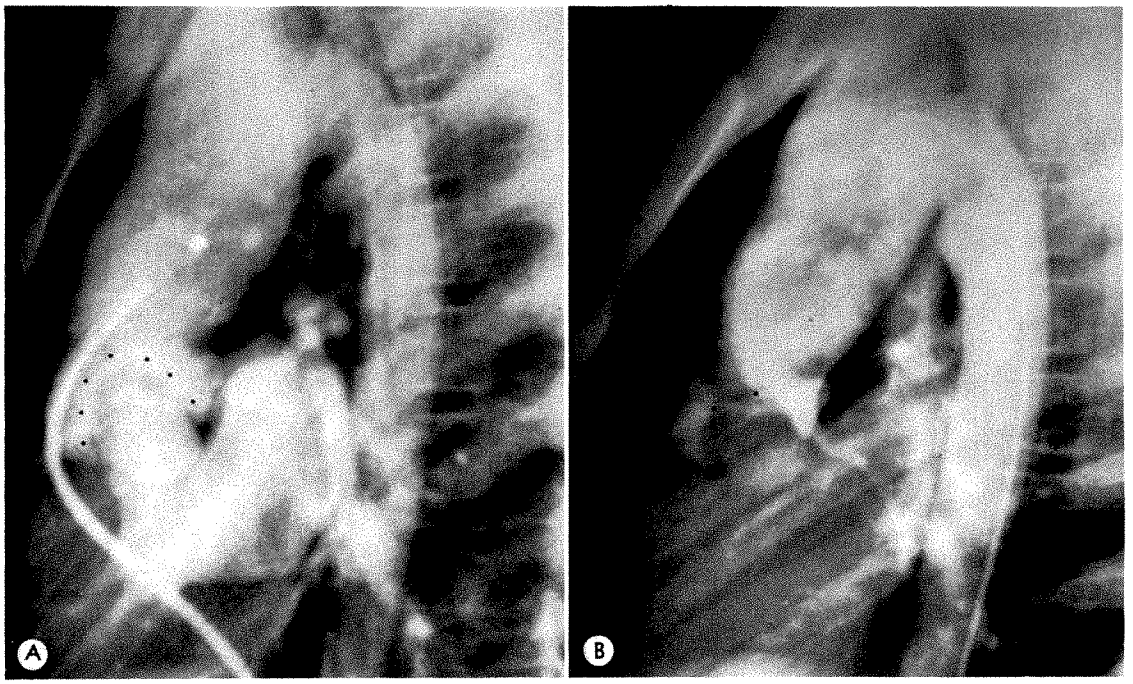


FIG. 3. Case 9. (*A*) Lateral projection during pulmonary artery angiocardiography in 9 year old girl with congenital aortic valve stenosis. Doming of thickened aortic valve leaflets is demonstrated during ventricular systole, and there is poststenotic dilatation of the ascending aorta. The examination excludes subvalvar and supravalvar aortic stenosis and other cardiac abnormalities. However, it fails to provide information regarding the severity of the aortic valve stenosis. (*B*) Left lateral projection during thoracic aortography immediately following the pulmonary artery angiocardiogram in the same patient. The domed aortic valve leaflets are again demonstrated, and the area of the radiolucent jet is 8 per cent of the area of the aorta, indicating severe aortic stenosis.

tion) of the aorta at the level of the transition between the sinuses and ascending portion is present (Fig. 1 and 5, inclusive).

According to our findings, severe aortic stenosis is present when the area of the valve ostium, as calculated from the diameter of the jet in one projection, is less than 10 per cent of the area of the aorta, and surgery is indicated when the area of the ostium is reduced to 15 per cent or less of the area of the aorta. These findings tend to confirm the statements of others that the area of the valve orifice must be reduced to at least one-fourth normal before the stenosis assumes hemodynamic significance,[63] and that the "critical" orifice area in adults with pure stenosis is 0.5 cm.^{2}.[29] They are further substantiated by studies of pressure gradients and flow rates across experimentally produced narrowings of the proximal aorta[34] and renal artery[49] and have been confirmed by observations in an experimental model[22] and autopsy material.[50]

In spite of the great amount of information about the aortic valve provided by thoracic aortography, the technique has certain inadequacies. It cannot exclude hypertrophy of the left ventricular outflow tract or survey the heart for additional abnormalities. Therefore, right heart catheterization and pulmonary artery angiocardiography frequently are indicated as complementary procedures. It demonstrates the jet in only one projection, probably in its shorter diameter. Since the valve orifice may have a fish-mouth configuration, the actual area of the opening may differ to some extent from the area calculated on the basis of the diameter measured in only one plane. In addition, the radiolucent jet cannot be precisely defined in some patients, even when other changes of aortic stenosis, including marked doming of valve leaflets into the aorta are classically demonstrated (Fig. 5). When the jet is not distinct, other methods of determining the severity of stenosis must be used.

An insufficient number of patients have been studied to correlate adequately the size of the valve ostium, as demonstrated by the aortic jet, with the systolic pressure gradient across the valve, as determined by transvalvar left ventricular catheterization. It is important to bear in mind that the valve leaflets may be markedly deformed and/or the ostium considerably reduced in area in the absence of a hemodynamically significant pressure gradient or reduction in flow rate across the valve.



Fig. 4. Case 5. Thoracic aortogram in an 11 year old boy with congenital aortic valve stenosis. The aortic valve leaflets dome markedly into the aorta. The area of the valve ostium, calculated from the diameter of the radiolucent jet, is 5 per cent of the aortic area. There is poststenotic dilatation of the ascending aorta and whirling and eddying of blood in the ascending aorta, mixing with the contrast material. The left coronary arteries are predominant.

DISCUSSION

In the preoperative evaluation of patients with aortic valve stenosis, cardiac catheterization and angiography are necessary (1) to confirm the diagnosis and es-

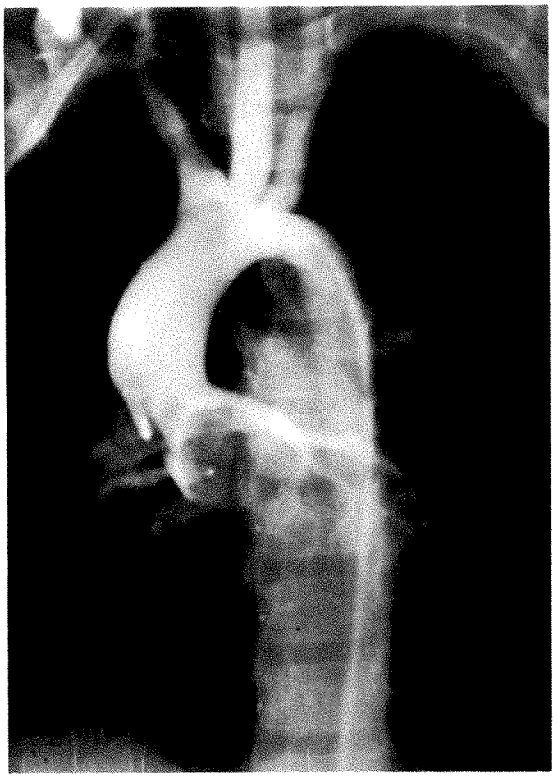


FIG. 5. Thoracic aortogram in an $8\frac{1}{2}$ year old boy with congenital aortic valve stenosis. The patient is in a slight right posterior oblique position. In spite of marked doming of aortic valve leaflets, waist formation at the base of the aorta, and poststenotic dilatation of the ascending aorta, a distinct aortic jet is not demonstrated.

pecially to investigate the possible presence of supravalvar aortic stenosis or hypertrophy of the left ventricular outflow tract, (2) to exclude additional or unsuspected cardiac abnormalities, and (3) to determine the severity of the stenosis.

Obtaining a pressure gradient across the aortic valve to determine the severity of stenosis may involve a high risk or be impossible in some patients. Even when obtained, it may not accurately indicate the severity of the stenosis. Therefore, we have sought an angiographic technique complementary to catheterization which would make possible safe and accurate preoperative evaluation of patients with clinically suspected aortic valve stenosis. We believe right heart catheterization with pulmonary artery angiocardiography is the safest and most effective method of satisfactorily accomplishing the first two requirements cited above, and, of the currently utilized angiocardiographic techniques, thoracic aortography provides the best means of determining the severity of the stenosis. Therefore, we have evolved the following program for preoperative evaluation of patients with aortic stenosis: (1) right heart catheterization and pulmonary artery angiocardiography to rule out hypertrophy of the left ventricular outflow tract and supravalvar aortic stenosis and to exclude additional or unsuspected cardiac abnormalities, and (2) retrograde catheterization of the left ventricle through the aortic valve, if possible, to obtain a pressure gradient, followed by thoracic aortography to confirm the diagnosis of aortic stenosis and estimate its severity. We believe more hazardous catheterization and angiocardiography techniques are not justified.

The above plan has the advantage that in patients with minimal stenosis, the left ventricle may be catheterized through the aortic valve with relative ease; and in patients with severe stenosis in whom the valve cannot be passed with a catheter, aortography may be done to estimate the severity of the stenosis by direct measurement of the ostium. The aortic jets which are most distinct and most amenable to accurate calculation are most likely to be demonstrated in patients with severe degrees of stenosis in whom retrograde catheterization of the left ventricle for a pressure gradient is most difficult. In our series, this diagnostic approach was most beneficial to those patients (Cases 2, 4, 5, and 8) with severe stenosis in whom transvalvar left ventricular catheterization was impossible. The area of the ostium in these patients was calculated to be less than 9 per cent of the aortic area; corrective surgery was performed without first obtaining a pressure gradient to determine the degree of stenosis, and in all cases severe stenosis was found at operation. In those patients in whom the ostium area was 9–15 per cent of the aortic area, unequivocal but rela-

tively less severe stenosis was found at surgery, and only minimal valvotomy was performed in order not to create iatrogenic aortic insufficiency.

SUMMARY

In 22 patients with aortic valve stenosis, the size of the valve ostium has been calculated by measuring the base of the radiolucent jet produced by ejection of nonopaque blood from the left ventricle into the opacified aorta during thoracic aortography. In the preoperative evaluation of patients for aortic valve surgery, the severity of aortic stenosis may be estimated by this angiographic technique. It is considered a complementary procedure to right heart catheterization, pulmonary artery angiocardiography, and, if possible, transvalvar catheterization of the left ventricle for purposes of obtaining a systolic pressure gradient across the valve. The technique is particularly useful in patients with severe stenosis in whom transvalvar catheterization of the left ventricle is impossible.

Severe aortic stenosis is present when the area of the ostium is less than 10 per cent of the aortic area. In these patients, corrective surgery is believed to be indicated without requiring preoperatively a transvalvar pressure gradient to determine the severity of the stenosis. Surgically treatable but less severe aortic stenosis is present when the area of the ostium is 10–15 per cent of the aortic area. Further studies are necessary to determine the hemodynamic significance of an ostium area 16–25 per cent of the aortic area.

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A NEW VASCULAR INJECTOR FOR ANGIOGRAPHY

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FROM my experience with direct percutaneous noncatheter brachial angiography, it was apparent that a more satisfactory injector was desired.[1] This injector was designed with particular emphasis being placed on the ease of performance of brachial angiography, and at the same time improvement of other methods of angiography.* Two of the main requirements were simplicity of design and the development of a more uniform back up pressure.

The injector is lightweight (47 pounds)

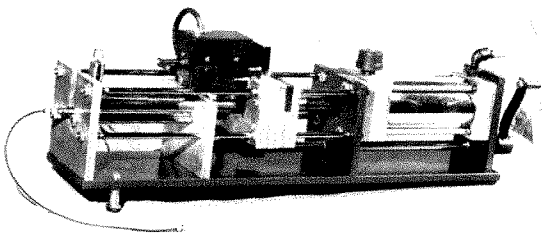


FIG. 1. The Marshall automatic vascular injector. The automatic timing device, which can be seen in place, is designed so that it may be added or removed easily from the injector.

and may be transported easily from one hospital to another (Fig. 1); it can be placed on the side of the table and both the patient and the injector can be moved, permitting roentgenography of multiple areas from a single brachial artery injection or from a transfemoral catheter injection. There are very few parts in the injector in which mechanical trouble might occur, thereby giving the operator more confidence in its use. By the placement of a reducing gear on the filling crank, the injector can be employed to perform lymphangiography.

* Sigmamotor Co., Inc., 10 North Main Street, Middleport, New York.

DESCRIPTION OF INJECTOR

The power with which to drive the metal syringe is obtained from a tank of compressed nitrogen (compressed air and nitrous oxide may be used). A quick disconnect coupling is present on the regulator end of the injector gas hose. This opens and closes by sliding the collar away from the hose. A bleeder valve is present to decompress the gas hose after turning off both tank valves and before attempting to disconnect the injector hose from the gas regulator.

It is important to know the difference between gauge pressure and hydraulic pressure in an injection system. The relationship may be expressed mathematically as follows:

$P_1 \times A_1 / A_2 = P_2$ (hydraulic pressure), where P_1 is the applied gauge pressure,

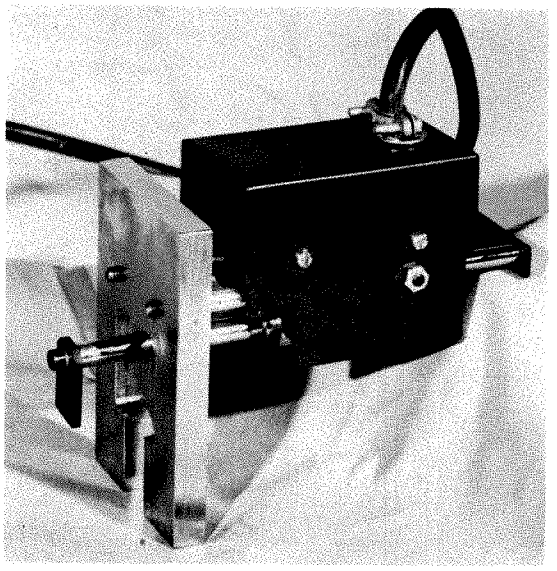


FIG. 2. Close-up view of the automatic timing device which permits a wide range of film exposures.

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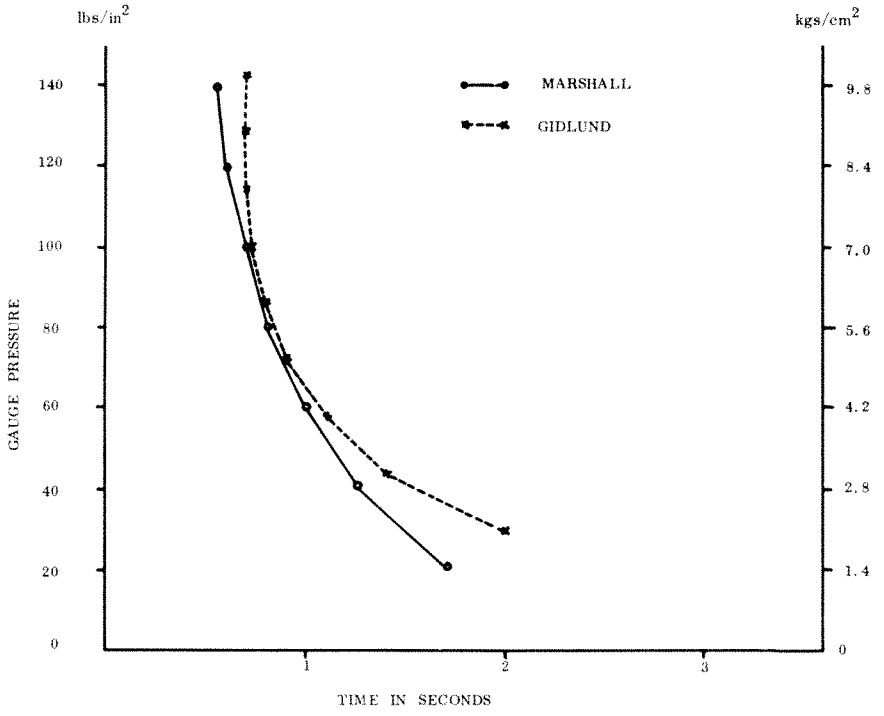


FIG. 3. Graphs obtained with the ·——· Marshall and the *— —* Gidlund injectors: No. 4 (gray) Ödman-Ledin connecting catheter (46 cm.); 30 cc. meglumine iothalamate, 60 per cent.

$A1$ area of the face of the metal (air piston), $A2$ area of the syringe plunger face, and $A1/A2$ the mechanical advantage of injector.

The diameter of the compression chamber piston is $3\frac{1}{4}$ inches, and the diameter of the syringe plunger face is $1\frac{5}{8}$ inches, therefore

$$\frac{A1}{A2}=\frac{3.14\times r^2}{3.14\times r^2}=\frac{3.14\times\left(\frac{6.5}{(4)}\right)^2}{3.14\times\left(\frac{6.5}{(8)}\right)^2}$$

$=4$ to 1 (mechanical advantage).

The desired pressure in the pressure cylinder is regulated by a two stage reducing valve. The pressure is indicated in pounds per square inch on a manometer. This injector permits an extremely fast delivery time of the contrast medium which is essential in obtaining a good bolus in performing brachial angiography. Nitrogen is used as the gas to drive the compression cylinder because of the nonexplosive nature and the fact that a more uniform distribution of the pressure in the tank remains after several injections have been made. A size H nitrogen cylinder is usually employed and preferred.

The compression chamber has a diameter of $3\frac{1}{4}$ inches and a stroke of 5 inches. The maximum safe pounds per square inch of applied pressure to the cylinder is 250 p.s.i. air. The chamber is of durable construction to ensure a long life.

The stainless steel syringe has a volume of 150 cc. and multiple injections of any amount may be made without having to stop and reload the syringe. Air is removed from the top of the syringe and the distal portion of the syringe is elevated 15 degrees to aid in the removal of the air. The stroke is adjusted so that 20 cc. of contrast medium remains in the syringe to stop any accidental air injection. This is the effective dead space. After the injection, the syringe can be reloaded with contrast

medium through a plastic tubing and 3-way stopcock. This type of reloading has proven to be simple and fast. A simple heating jacket is applied around the syringe to warm the contrast material to body temperature or the material may be warmed prior to placing it in the syringe.

A simple electronic controlled timing switch is used to correlate the injection of the contrast medium with film exposure (Fig. 2). This permits a wide range of film exposure from before, during and after the injection of the contrast medium. This automatic timing circuit is so designed that it may be added or removed very easily from the injector. For a brachial injection a gauge pressure between 90–100 p.s.i. is used and for a Seldinger renal arteriography a 65–90 p.s.i. gauge pressure.

A comparison of this injector with the Gidlund automatic injector can be seen in Figures 3 and 4. The Gidlund automatic pressure injector does not permit multiple injections without stopping to reload the syringe.

SUMMARY OF IMPORTANT FEATURES OF DESIGN

1. The injector has a pressure control on the gas cylinder in p.s.i. (pounds per square inch) with a range from 0 to 200 pounds per square inch. This permits hydraulic (injection) pressures up to 800 p.s.i. The highest (hydraulic) used in angiography at the present time is 450 p.s.i. By changing the reducing valve, even larger pressures may be developed.

2. The size of the device is small so that it may be placed on the table top and moved with the patient. The injector has a total weight of 47 pounds.

3. Designed especially for brachial angiography, it can be used for all types of angiocardiography and arteriography.

4. An automatic timing device correlates injection and roentgenography.

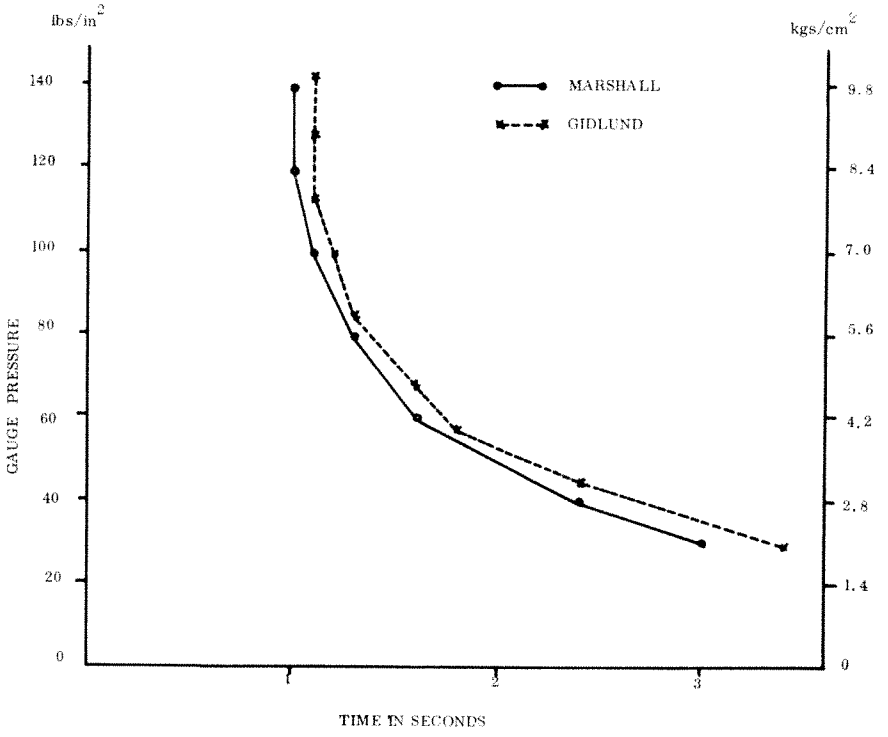


FIG. 4. Graphs obtained with the Marshall and the Gidlund injectors: No. 3 (yellow) Seldinger catheter (88 cm.); 20 cc. sodium iothalamate, 80 per cent.

5. Distant control of the start of injection is possible.

6. Multiple injections of different amounts may be made without reloading the syringe.

7. A mechanical advantage of 4 to 1 is obtained.

8. Very rapid injection times can be had and a good back up pressure is obtained.

9. The design is simple with very few moving parts. This decreases the chance of mechanical failure during the injection.

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APPLICATION OF THE POLAROID-LAND METHOD OF ROENTGENOGRAPHY TO ANGIOCARDIOGRAPHY

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THE achievement of satisfactory results in large-film selective angiocardiography depends on many factors. These are: (1) the choice of injection site, (2) the delivery of an optimal dose of contrast medium per unit time, (3) a properly selected filming sequence, and (4) the careful control of roentgenographic technique by those well trained in the basic principles of roentgenography.

Of prime importance, not only from the standpoint of obtaining a rewarding study but also from the standpoint of the safety of the procedure, is the careful positioning of the catheter tip preparatory to the injection of contrast medium. It must be determined that the catheter tip lies in the proper portion of the chamber into which the injection is to be made and that the tip does not lie in such a position that a jet of contrast medium emanating from the catheter is likely to be trapped beneath a trabecular muscle. Some unfortunate "accidents" that have occurred during angiocardiography have undoubtedly resulted directly from lack of care in determining the proper and free position of the catheter tip immediately prior to injection.

Although the injection of small amounts of contrast medium through the catheter and observation of this injection fluoroscopically may provide accurate information in some cases, it is frequently desirable to view the catheter in both the frontal and lateral projections. The position of the catheter in the frontal projection is readily determined by fluoroscopy using the image intensifier employed for cardiac catheterization. Assessment of catheter position in the lateral projection is a more difficult problem which, in the past, has been solved either by the use of a hand fluoroscope or by a conventional spot-film technique employing the lateral angiographic roentgenographic tube as the source of radiation. There are obvious disadvantages to each of these techniques. The use of a conventional spot-film technique imposes a waiting period of approximately 10 minutes for processing of the film, and, if one or more attempts at repositioning the catheter are made, an additional 10 minute waiting period is necessary for the processing of each additional spot film. The use of a hand fluoroscope requires dark adaptation and results in a considerable amount of radiation to the patient and to the fluoroscopist.

In the search for a more rapid and satisfactory method for assessing catheter position in the lateral projection, 2 years ago we decided to investigate the possible use of the Polaroid-Land method of roentgenography for this purpose. A Polaroid roentgenographic machine, which had been in use in the Section of Radiology for several years and which had been employed previously with success in the assessment of needle position in aortography and certain neurosurgical procedures, was utilized for this investigation.

This paper describes briefly the technique of Polaroid roentgenography as we have employed it for catheter localization in 300 angiocardiographic examinations. This useful application of Polaroid roentgenography has not been previously reported.

METHOD AND TECHNIQUE

A new one-step, essentially dry photographic process was first described by Land in 1947. The roentgenographic application of the method was subsequently developed utilizing the technical facilities of the

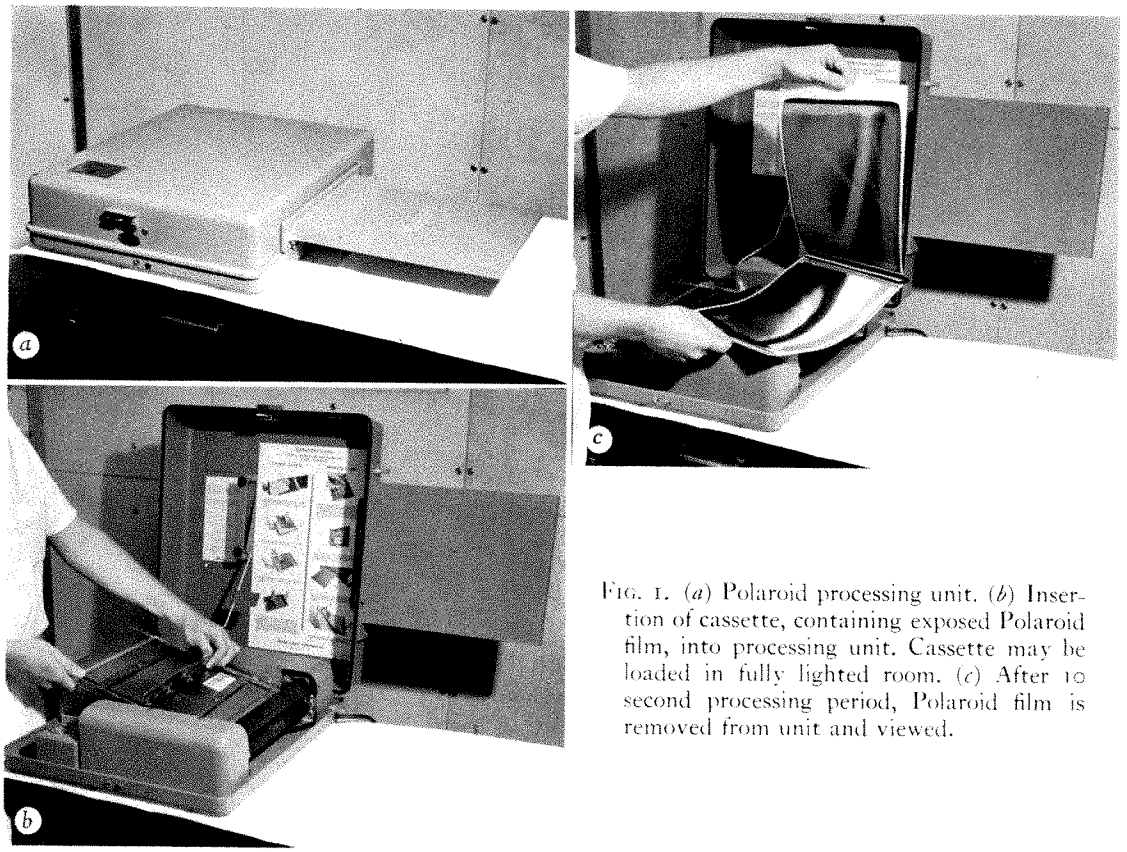


FIG. 1. (*a*) Polaroid processing unit. (*b*) Insertion of cassette, containing exposed Polaroid film, into processing unit. Cassette may be loaded in fully lighted room. (*c*) After 10 second processing period, Polaroid film is removed from unit and viewed.

Polaroid Corporation and the roentgenologic facilities of the Massachusetts General Hospital. In more recent years, this method of roentgenography has been further perfected and now finds a number of important applications in diagnostic radiology.

The advantages of this roentgenographic method lie primarily in the rapidity with which the film can be processed and viewed. The new type of Polaroid film requires only 10 seconds for processing, and the loading of cassettes and processing may be carried out in a fully lighted room. The Polaroid developing unit is small and portable and may be conveniently plugged into any 110 volt AC electrical outlet (Fig. 1, *a*, *b* and *c*).

For lateral chest roentgenography to determine catheter position just prior to angiocardiography, the following procedure is employed. When the diagnostic catheterization procedure has been completed, the angiocardiographic catheter is placed in what is believed to be the desired site of injection as observed on the image intensifier fluoroscope. The table top is moved cephalad and the patient's thorax is posi-

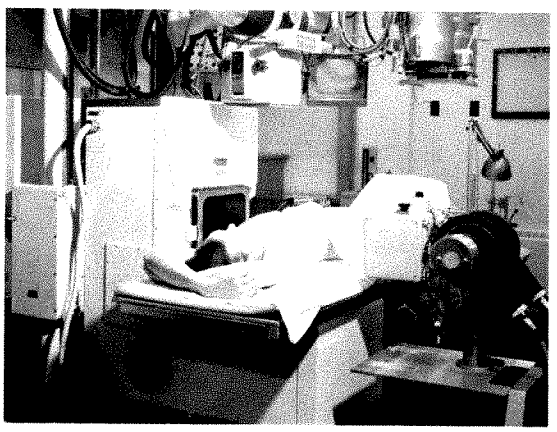


FIG. 2. Patient and equipment in position for biplane angiocardiography. Loaded Polaroid cassette is placed in front of the lateral aperture of the film changer. Film exposure is made utilizing horizontally directed roentgenographic tube.

tioned over the biplane film changer for angiocardiography. The loaded Polaroid grid cassette with high-speed screens is placed between the patient's thorax and the lateral aperture of the film changer. The lateral (horizontally directed) roentgenographic tube utilized for angiocardiography is moved into place and serves as the source of radiation (Fig. 2).

Scout roentgenograms made at the time of the patient's arrival in the catheterization-angiocardiography room serve as a convenient guide to proper exposure of the Polaroid film. When using the Polaroid grid cassette with high-speed screens, we have found that approximately one-fifth to

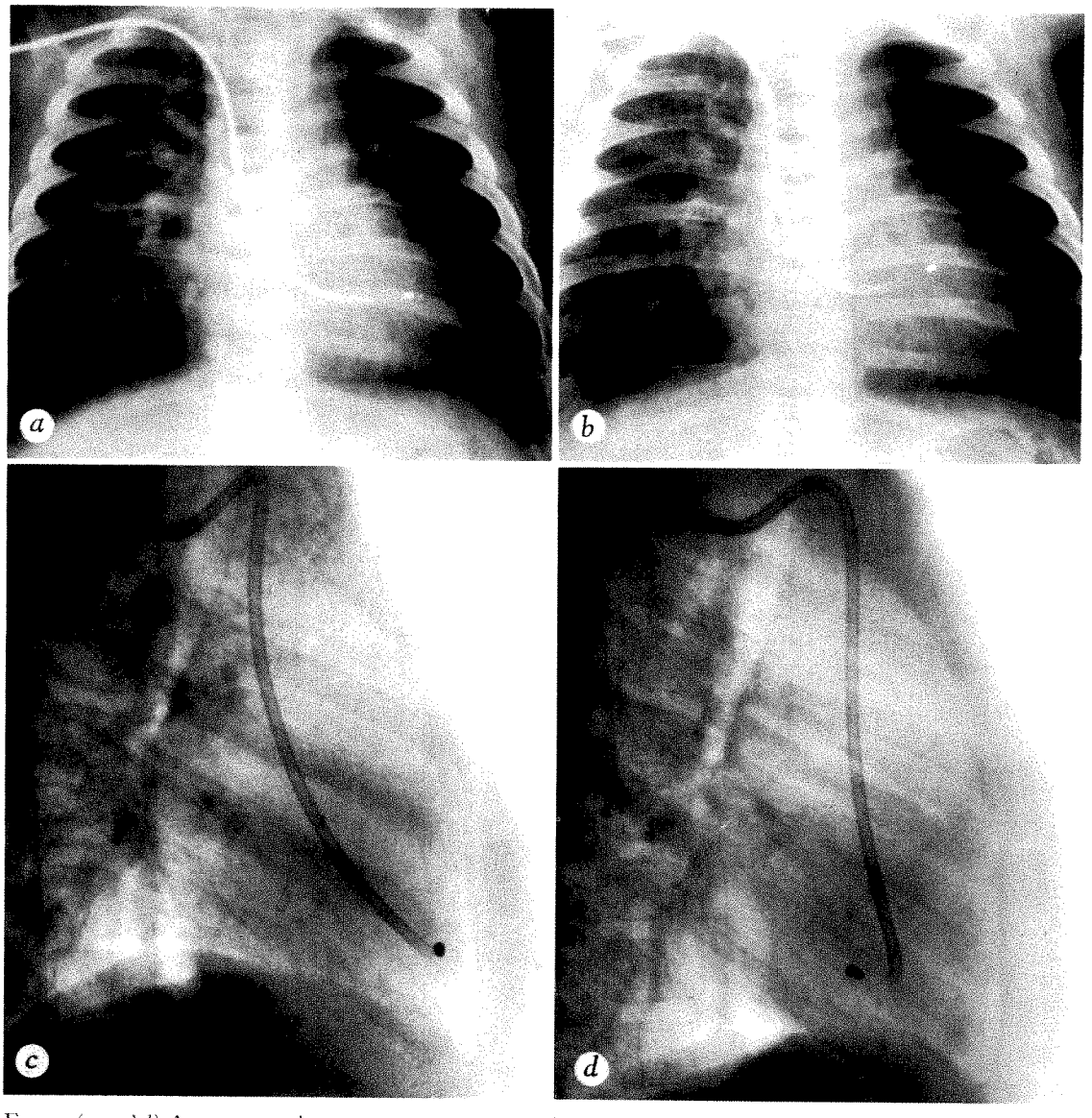


FIG. 3. (*a* and *b*) Anteroposterior spot roentgenograms showing angiocardiographic catheter in right ventricle (*a*) and in left ventricle (*b*). Note similar positions of catheter in this projection. In this case, the pressures within the two ventricles were equal and it was impossible, without viewing the catheter in the lateral projection, to know within which ventricle the catheter tip lay. (*c* and *d*) Lateral Polaroid roentgenograms showing catheter tip in right ventricle (*c*) and in left ventricle (*d*). In the latter instance, the catheter tip had passed from the right ventricle to the left ventricle through a ventricular septal defect.

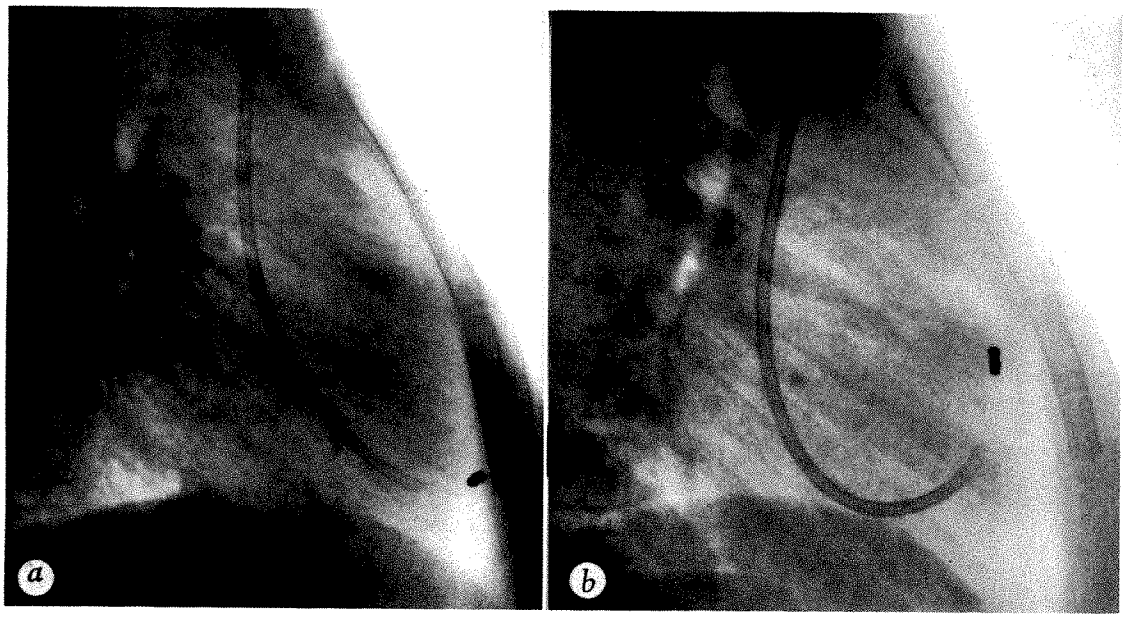


Fig. 4. (*a*) Lateral Polaroid roentgenogram showing poor position of catheter in right ventricle. Catheter tip is directed against the anterior wall of the right ventricle and possibly extends beneath a trabecular muscle. (*b*) Catheter in satisfactory position in right ventricle with tip free in the region of the ostium infundibulum and directed toward the pulmonary valve.

one-third of the exposure used for an optimally exposed scout film will result in a properly exposed Polaroid film. This exposure adjustment may be made by regulation of the milliampere seconds or kilovoltage, or both.

Following proper selection of the roentgenographic factors, the Polaroid film is exposed, developed, and viewed immediately. If the catheter tip is found to be properly positioned within the desired cardiac chamber, the angiocardiographic examination is then carried out (Fig. 3, *a*, *b*, *c* and *d*). Should the catheter position not be satisfactory, the catheter is redirected and another Polaroid film may be exposed (Fig. 4, *a* and *b*).

SUMMARY AND CONCLUSIONS

Assessment of catheter position prior to selective angiocardiography in many instances may be made accurately only by viewing the catheter in both the frontal and lateral projections. While the position of the catheter tip is easily determined in the frontal projection by means of fluoroscopy, visualization of the catheter in the lateral projection presents a more difficult problem. This problem heretofore usually has been solved by means of lateral spot roentgenograms or by the use of a hand fluoroscope. Both of these methods have serious disadvantages. The Polaroid-Land method of roentgenography provides a simple, convenient, and rapid method of catheter localization in the lateral projection with a minimum of radiation to the patient and to the physician. This previously undescribed application of Polaroid roentgenography has produced rewarding results when used at the Mayo Clinic in 300 angiocardiographic examinations.

Mayo Clinic
Rochester, Minnesota

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A NEW ILLUMINATOR FOR COPYING ROENTGENOGRAMS*

By J. T. LITTLETON, M.D., *and* H. C. REYNOLDS, B.S.
SAYRE, PENNSYLVANIA

THE wide variations in translucency of roentgenograms make them difficult to copy by any photographic method without some equalization of contrast. This may be accomplished by providing additional light in dark spots or diminishing light in the more translucent areas. The latter may

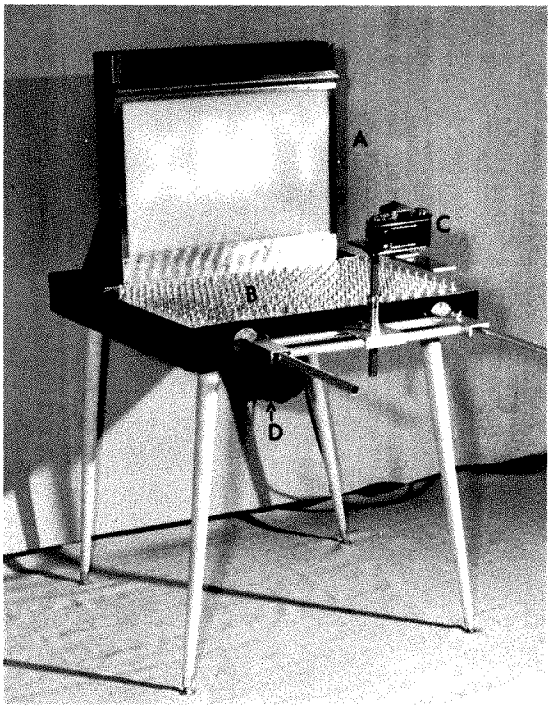


FIG. 1. Illuminator with "XRAY" spelled out on viewing panel (A) to illustrate the small zone of illumination from each bulb; (B) resistor panel; (C) 35 mm. camera on adjustable stand; and (D) blower.

be accomplished by holding a paper cut to shape over the translucent areas of the film or by making an unsharp copy of the roentgenogram and using this as a filter. Both of these techniques consume significant amounts of time and photographic material and may cause false relief where significant contrast is present.

Lindblom[1] developed an apparatus for equalization of translucency by increasing the light in the dark spots. He constructed a viewbox containing 704 flashlight bulbs in an area 35×45 cm. The bulbs lying under dark spots on the roentgenogram were illuminated and raised or lowered so as to give an equal distribution of light over the film.

To circumvent some of the problems inherent in this type of contrast development, the authors have constructed another device which has a wide flexibility in the development of contrast between areas of varying translucency. When used with precise monitoring of light levels between comparative areas, very satisfactory reproductions can be obtained. The illuminator

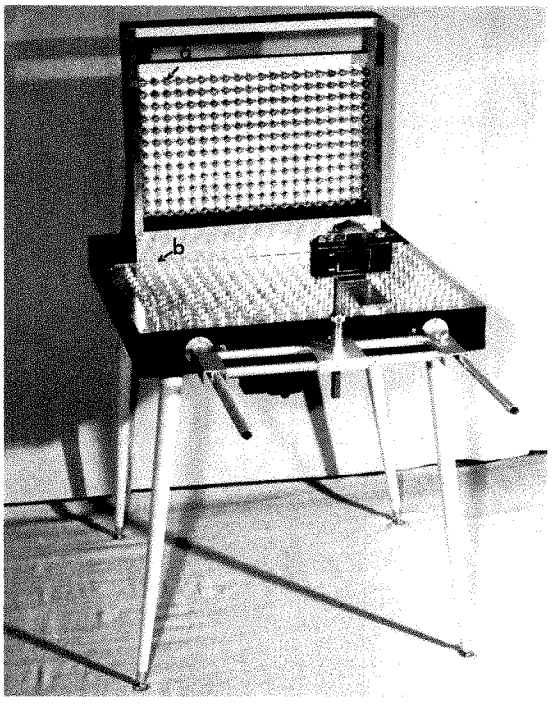


FIG. 2. Opal viewing panel removed to show bulbs (a) and resistor knobs (b).

* From the Department of Radiology of the Guthrie Clinic Ltd. and Robert Packer Hospital, Sayre, Pennsylvania.

(Fig. 1) has an illuminated surface 12 by 20 inches with a light source every square inch for a total of 240 light sources which develop a total illumination of 1,200 watts. The intensity of each bulb is varied from 0 to 5 watts by a variable 20 ohm resistor. The panel is energized from a 12 volt transformer, and further control of light intensity can be effected by putting a variable resistance in the line to the transformer. An old portable roentgen ray control unit performs this function very well. The 20 ohm resistors and the large bank of 5 watt bulbs develop a significant amount of heat which is dissipated by a 140 cubic foot blower beneath the panel of resistors with an exit past the bulb panel (Fig. 2). This entire unit was constructed in a home workshop at an approximate materials cost of $500. The parts used in this particular unit are as follows:

Sockets:	Leecraft Manufacturing Company, Inc. 21–16 44th Road Long Island 1, N. Y.	Part No. 11-207-24 with 24″ leads
Control:	Centralab Milwaukee 1, Wisconsin	WN-200-20 Ohms Linear Taper
Lamps:	General Electric Co.	Type No. 89
Transformer:	General Electric Co.	Model 9T56Y2884 KVA 1.5 Voltage 120-12
Blower:	Dayton Electric Manufacturing Co. Chicago, Illinois	Model 2060 Capacity—140 cu. ft.
Light Meter:	Science and Mechanics Kit and Product Division 450 East Ohio Street Chicago 11, Illinois	Model 250P

The actual copying of the roentgenograms can be done with significant ease and consistent reproducibility. The roentgenogram to be copied is placed on the viewing surface and suitably masked. Light contrast is developed by raising and lowering illumination by means of the panel of resistors. The light levels of comparable areas can be precisely adjusted and monitored over a very small area with a sensitive light meter which is provided with a photocell having an 0.5 inch aperture. For rapid copy, a polaroid camera provides a very suitable reproduction in either a transparency for projection or a simple paper positive. For projection to large audiences, very satisfactory slides have been made with 35 mm. direct positive film. If available, a second 35 mm. camera containing negative film can be used at the same time to provide negatives for subsequent printing to either transparency or paper positives.

The authors have found that this copying system provides significant flexibility not only in the development of contrast, which can be provided by other systems including a logEtronic system,[2] but in addition it permits the development of photographic emphasis to a specific area which cannot be accomplished by other techniques. Figure 3, *A*, *B* and *C* shows the effect of contrast enhancement which can be developed with selective illumination. We have found that it has been relatively

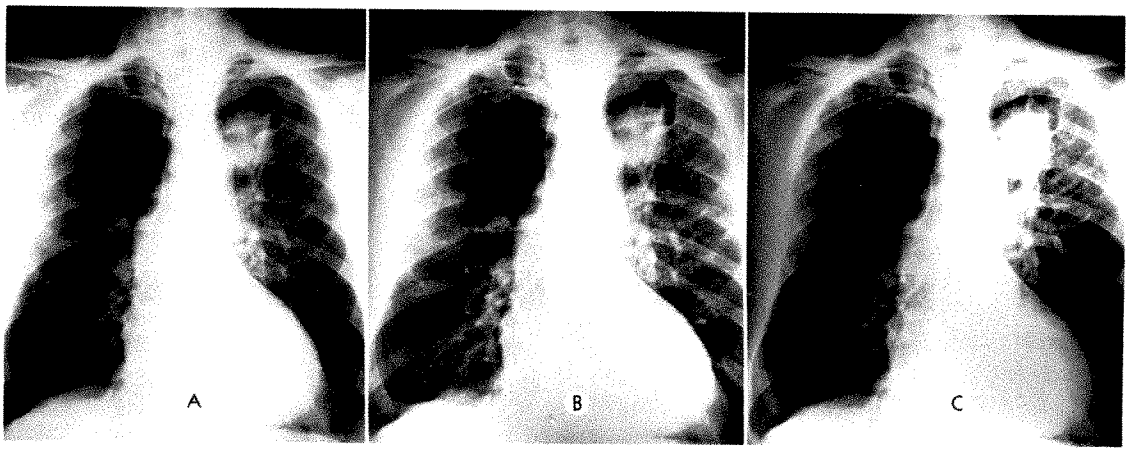


FIG. 3. (*A*) Overpenetrated roentgenogram copied with flat illumination from Picker high intensity illuminator. (*B*) Same roentgenogram with balanced illumination. (*C*) Illumination adjusted to emphasize lesion in the left upper lobe.

simple to instruct technicians in the use of this apparatus.

SUMMARY

A new illuminator for copying roentgenograms is described. Selective illumination permits the development of significant contrast in reproductions, circumvents the shortcomings of photographic techniques to accomplish this end and permits one to emphasize the area of primary interest by selective illumination. The entire device can be constructed in a home workshop at a parts cost of approximately $500.

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DOUBLE BLIND CLINICAL COMPARISON OF CONRAY WITH RENOGRAFIN AND HYPAQUE IN PERIPHERAL ANGIOGRAPHY*

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CONRAY 60 per cent* is a new iodine-containing contrast agent recently introduced for angiography and intravenous urography. During the first months of its clinical use numerous papers have indicated that it is a safe and effective contrast material for excretory urography,[6,11,12] cerebral angiography,[1,4,6,7,8,10,11,12] aortography,[6,12] and peripheral angiography.[6,11,12] All of these reports, however, have simply evaluated the use of conray in a series of patients and have not compared it clinically with other contrast agents currently utilized for similar purposes. Thus, the tolerance of patients to conray relative to other angiographic contrast materials is somewhat uncertain. The purpose of this paper is to report the findings in a double blind clinical comparison of conray with two widely used intravascular contrast agents, renografin† and hypaque,‡ in patients having intra-arterial and intravenous peripheral angiography, respectively.

PROPERTIES OF CONRAY

Conray (meglumine iothalamate) 60 per cent is a sterile aqueous solution containing 60 per cent w/v of the N-methylglucamine salt of 5 acetamido-2,4,6-triiodo-N-methylisophthalamic acid (Fig. 1). Each milliliter contains 0.6 gm. of meglumine iothalamate to which are added stabilizing and buffering agents. The free acid contains 62 per cent iodine, and the 60 per cent solution provides 28.2 per cent iodine (282 mg./ml.).

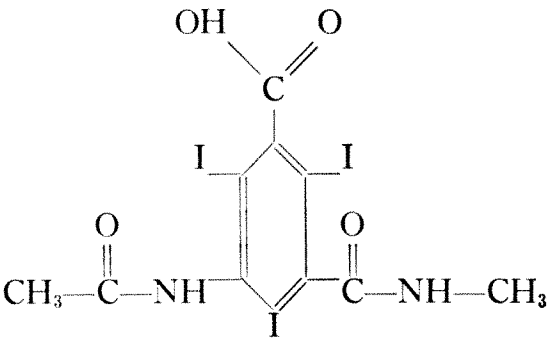


FIG. 1. Chemical formula of conray (iothalamate). Molecular weight 614.

The iodine content and viscosity of conray are shown in Table I and compared with those of renografin (methylglucamine diatrizoate) and hypaque (sodium diatrizoate).[5]

MATERIAL AND METHODS

A total of 460 consecutive adult patients included in the study had 502 examinations and 755 injections of contrast material, 499 intra-arterial and 256 intravenous. Conray was compared with renografin in 3 intra-arterial and with hypaque in 3 intravenous peripheral angiographic procedures (Table II). Two hundred and seventy-six patients were men and 184 were women. Table III shows the ages of the patients in decades according to the type of examination performed.

No attempt was made to evaluate the tolerance of the intracranial central nervous system to the direct intravascular injection of the contrast agents being tested. Therefore, *excluded* from the study were those procedures which require injections in the brachial artery, thoracic aorta,

* Mallinckrodt Pharmaceuticals, Mallinckrodt Chemical Works, St. Louis 7, Missouri.

† E. R. Squibb, and Sons, 745 Fifth Avenue, New York 23, New York.

‡ Winthrop Laboratories, 1450 Broadway, New York 18, New York.

* From the Edward Mallinckrodt Institute of Radiology, Washington University School of Medicine, St. Louis, Missouri.

TABLE I

COMPARISON OF IODINE CONTENT AND VISCOSITY OF CONRAY, RENOGRAFIN AND HYPAQUE

	Conc. w/v	Iodine Content gm./100 ml.	Viscosity	
			25° C.	37.5° C.
Conray (meglumine iothalamate)	60	28.2	6.1	4.0
Renografin (methylglucamine diatrizoate)	60	29.3	6.2	4.1
Hypaque (sodium diatrizoate)	50	29.9	3.5	2.5

or heart, and, thereby, expose the intracranial contents to high concentrations or volumes of contrast material. Also excluded from the study were all children, some of whom had examinations under general anesthesia. The only patients who might have qualified for conray but who did not receive it were those referred by one surgeon for aortography to evaluate the renal arteries and kidneys. He preferred not to have an "experimental" contrast agent used in his patients. This explains the relatively larger number of patients in whom renografin was used for retrograde abdominal aortography.

All adult patients referred for any one of the six different peripheral angiographic procedures included in the study were assigned a contrast material according to their roentgenologic examination numbers. Thus, conray was used in all patients with odd numbers and either renografin or

TABLE II

EXAMINATIONS PERFORMED AND INJECTIONS MADE IN 460 PATIENTS HAVING PERIPHERAL ANGIOGRAPHY

	Renografin		Conray		Hypaque		Total	
	Examinations	Injections	Examinations	Injections	Examinations	Injections	Examinations	Injections
Intra-arterial								
1. Femoral arteriography	46	57	48	59	—	—	94	116
2. Aorto-ilio-femoral arteriography	85	125	87	122	—	—	172	247
3. Retrograde catheter abdominal aortography	72	92	33	44	—	—	105	136
							371	499
Intravenous								
4. Bilateral pelvic phlebography with inferior vena cavagraphy	—	—	49	102	57	117	106	219
5. Lower extremity phlebography	—	—	7	12	11	17	18	29
6. Upper extremity and/or superior vena cavagraphy	—	—	2	2	5	6	7	8
							131	256
	203	274	226	341	73	140	502*	755

* The total number of examinations performed exceeds the number of patients studied because some patients had more than one examination.

TABLE III

NUMBER OF PATIENTS ACCORDING TO AGE IN DECADES AND TYPE OF EXAMINATION PERFORMED

	10–19	20–29	30–39	40–49	50–59	60–69	70–79	80–89	Total
Femoral arteriography	2	2	4	11	20	28	21	6	94
Aorto-ilio-femoral arteriography	0	1	5	25	56	51	28	6	172
Retrograde catheter abdominal aortography	7	8	25	23	18	16	8	0	105
Pelvic phlebography with inferior vena cavagraphy	6	9	16	20	25	22	7	1	106
Lower extremity phlebography	1	2	7	3	4	1	0	0	18
Upper extremity and/or superior vena cavagraphy	0	0	1	1	2	1	2	0	7
Total	16	22	58	83	125	119	66	13	502

hypaque was used in all patients with even numbers, depending upon whether they were to have an intra-arterial or an intravenous type of examination. The type of contrast material used was unknown to the radiologist and the patients during the examinations, and the patients were not aware that their reactions were being assessed in a comparative type of study. During the procedure observations were made of any serious reactions or complications, such as local extravasation of contrast material, dyspnea, hypotension, etc. The patient was then asked to indicate thoroughly what effect the injection had upon him. Every effort was made to avoid leading questions regarding the side effects listed in Tables IV and V.

TECHNIQUES OF EXAMINATION

Most patients were premedicated with 50 mg. each of diphenhydramine hydrochloride* and meperidine hydrochloride.† All arteriographic examinations and bilateral pelvic and inferior vena cavagraphy were done under 1 per cent lidocaine hydrochloride‡ local anesthesia.

Femoral arteriography was performed via percutaneous puncture of a common femoral artery with a 205 (or occasionally a 160) Seldinger needle. One or 2 injections of 35–50 ml. of 35–60 per cent conray or renografin were made, depending upon the size of the patient and the pathology under investigation. Most examinations required only a single injection of 35 ml. of 60 per cent contrast material.

Aorto-ilio-femoral arteriography was accomplished via 1 or 2 manual injections of 45–55 ml. of 60 per cent conray or renografin through a unilateral common femoral artery 205 Seldinger needle, with the patient performing the Valsalva maneuver.[2] Occasionally, aorto-ilio-femoral arteriography was either preceded or followed by femoral arteriography, in which case the maximum total amount of contrast material injected through the same femoral artery needle was 135 ml. of 60 per cent solution, in 3 separate injections. Ten patients (5 conray, 5 renografin) had retrograde abdominal aortographies via a mechanical injection of 50 ml. of 60 per cent contrast material directly through a unilateral common femoral artery needle, using a Gidlund pressure injector.

Retrograde abdominal aortography was performed via a red or yellow Ödman-Ledin catheter in the abdominal aorta introduced percutaneously through a common femoral

* Benadryl hydrochloride, Parke-Davis & Company, Detroit 31, Michigan.

† Demerol hydrochloride, Winthrop Laboratories, 1450 Broadway, New York 18, New York.

‡ Xylocaine hydrochloride, Astra Pharmaceutical Products Inc., Neponset Street, Worcester 6, Massachusetts.

TABLE IV

COMPARISON OF CONRAY AND RENOGRAFIN INTRA-ARTERIAL INJECTIONS

Injections	Femoral Arteriography				Aorto-Ilio-Femoral Arteriography				Catheter Abdominal Aortography				Total			
	Conray 59		Renografin 57		Conray 122		Renografin 125		Conray 44		Renografin 92		Conray 225		Renografin 274	
Side Effects	No.	Per Cent	No.	Per Cent	No.	Per Cent	No.	Per Cent	No.	Per Cent	No.	Per Cent	No.	Per Cent	No.	Per Cent
Warmth	33	56	24	42	69	57	60	48	34	77	70	76	136	60	154	56
Pain	31	53	31	55	66	54	65	52	13	30	22	24	110	49	118	43
Nausea	5	8.5	1	1.8	1	0.8	4	3.2	4	9.1	10	11	10	4.5	15	5.5
Vomiting	1	1.7	1	1.8	5	4.1	1	0.8	2	4.6	3	3.3	8	3.6	5	1.8
Bitter taste	—	—	—	—	1	0.8	2	1.6	—	—	—	—	1	0.4	2	0.7
Flush	—	—	—	—	—	—	2	1.6	1	2.3	—	—	1	0.4	2	0.7
Headache	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—
Chills	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—
Reactions																
Urticaria	1	1.7	—	—	1	0.8	1	0.8	—	—	—	—	2	0.9	1	0.4
Hypotension	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—
Dyspnea	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—
Phlebitis	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—
Arteritis	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—

Note: Percentages over 10 rounded to nearest whole number; percentages under 10 rounded to nearest tenth.

artery, using the Seldinger technique.[9] One or 2 injections of 25–60 ml. of 45–60 per cent conray or renografin were made.

Bilateral pelvic phlebography with inferior vena cavagraphy was performed via small polyethylene tubings introduced percutaneously into the common femoral veins bilaterally, using the Seldinger technique. Usually the total amount of contrast material injected was divided into 4 equal aliquots, and for each examination 2 bilateral simultaneous injections were made with roentgenography of the pelvis following 1 injection and the inferior vena cava following the other. Occasionally, a third injection was required to evaluate the pathology which was present more completely. Total dosage of conray was 90–180 ml. of 36–60 per cent contrast material, the usual dose being 100 ml. of 54 per cent or 120 ml. of

TABLE V

COMPARISON OF CONRAY AND HYPAQUE INTRAVENOUS INJECTIONS

Injections	Pelvic and Inferior Vena Cavagraphy				Lower Extremity Phlebography				Upper Extremity and Superior Vena Cavagraphy				Total			
	Conray 102		Hypaque 117		Conray 12		Hypaque 17		Conray 2		Hypaque 6		Conray 116		Hypaque 140	
Side Effects	No.	Per Cent	No.	Per Cent	No.	Per Cent	No.	Per Cent	No.	Per Cent	No.	Per Cent	No.	Per Cent	No.	Per Cent
Warmth	94	92	111	95	2	17	3	18	2	100	1	17	98	83	114	82
Pain	—	—	7	6	4	33	4	24	—	—	3	50	4	3.4	13	10
Nausea	4	3.9	3	3.9	1	8.3	1	6	1	50	1	17	6	5.2	5	3.6
Bitter taste	1	1.0	3	2.6	—	—	3	18	—	—	1	17	1	2.6	7	5
Vomiting	—	—	1	0.9	—	—	—	—	—	—	—	—	—	—	1	0.7
Flush	—	—	1	0.9	—	—	—	—	—	—	—	—	—	—	1	0.7
Headache	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—
Chills	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—
Reactions																
Urticaria	—	—	1	0.9	—	—	1	6	—	—	—	—	—	—	2	1.4
Hypotension	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—
Dyspnea	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—
Phlebitis	—	—	—	—	1	8.3	—	—	—	—	—	—	1	0.9	—	—
Arteritis	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—

Note: Percentages over 10 rounded to nearest whole number; percentages under 10 rounded to nearest tenth.

45 per cent. Total dosage of hypaque was 80–180 ml. of 35–50 per cent contrast material, the usual dose being 100 or 120 ml. of 37.5 per cent.

Lower extremity phlebography was accomplished by unilateral or bilateral injection of contrast material through a small percutaneous needle in a superficial vein over the dorsum of the foot with an ankle tourniquet in place and the patient in a 60 degree semi-erect position. When both lower extremities were studied by bilateral simultaneous injections, the procedure was tabulated as 2 separate injections and examinations. One or occasionally 2 injections of 30–50 ml. of 60 per cent conray or 50 per cent hypaque were made.

Upper extremity phlebography and/or superior vena cavagraphy was performed by unilateral or bilateral simultaneous injection of contrast material through 15–20 gauge antecubital vein needles. Dosage of conray 60 per cent was 40 ml. for a unilateral injection and 60 ml. in equally divided aliquots for bilateral simultaneous injections. Dosage of hypaque was 35–50 ml. of 35–50 per cent solution for unilateral injections and 90 ml. of 50 per cent solution in equally divided aliquots for bilateral simultaneous injections.

Examples of the opacification obtained with conray are shown in Figures 2 through 5.

RESULTS

In evaluating the tolerance of patients to the peripheral angiographic procedures performed, responses of the patients were divided into 3 categories, *i.e.*, side effects, drug reactions, and technical complications.

Side effects were arbitrarily defined as those self-limited and relatively benign responses of short duration which may follow the intravascular injection of an iodinated contrast material (Tables IV and V). Comparing intra-arterial conray with renografin and intravenous conray with hypaque, there were no significant differences between the frequencies of side effects (Tables IV and V).

Drug reactions were defined as the allergic, idiosyncratic or hypersensitivity responses of the patient which are potentially fatal and often require immediate therapy to prevent serious disability and even death. They include urticaria, hypotension which may lead to shock, and dyspnea which may result from laryngospasm, bronchospasm, or laryngeal edema. Urticaria developed in 2 patients following the injection of conray, in 2 patients following hypaque, and in 1 patient following renografin. None of the 755 injections, using any one of the three contrast agents being evaluated, was followed by any more severe drug reaction.

One patient developed erythema and warmth in a lower extremity 12–24 hours after the injection of 100 ml. of 60 per cent conray for phlebography. This disappeared spontaneously within 24 hours and was felt possibly to represent a chemical phlebitis. No other patient in this study had clinical evidence of arteritis or phlebitis following injection of contrast material.

Technical complications were defined as the mishaps which result from needle puncture or catheter placement, forceful injection of a bolus of alien material, intravascular manipulation of a foreign object, etc. These complications usually occur regardless of the type of contrast agent used. However, because some are associated with the extravascular injection of contrast material, all technical complications were tabulated in this study.

In a total of 502 examinations there were 8 technical complications, none of which was serious. They are listed in Table VI under the type of examination performed and with the contrast material indicated in parenthesis. Three patients developed hypotension during femoral artery puncture and before injection of contrast material. All rapidly responded to the intravenous injection of 0.5 ml. of methoxamine hydrochloride,* and the examinations were

* Vasoxyl, Burroughs Wellcome & Company, Inc., 1 Scarsdale Road, Tuckahoe 7, New York.

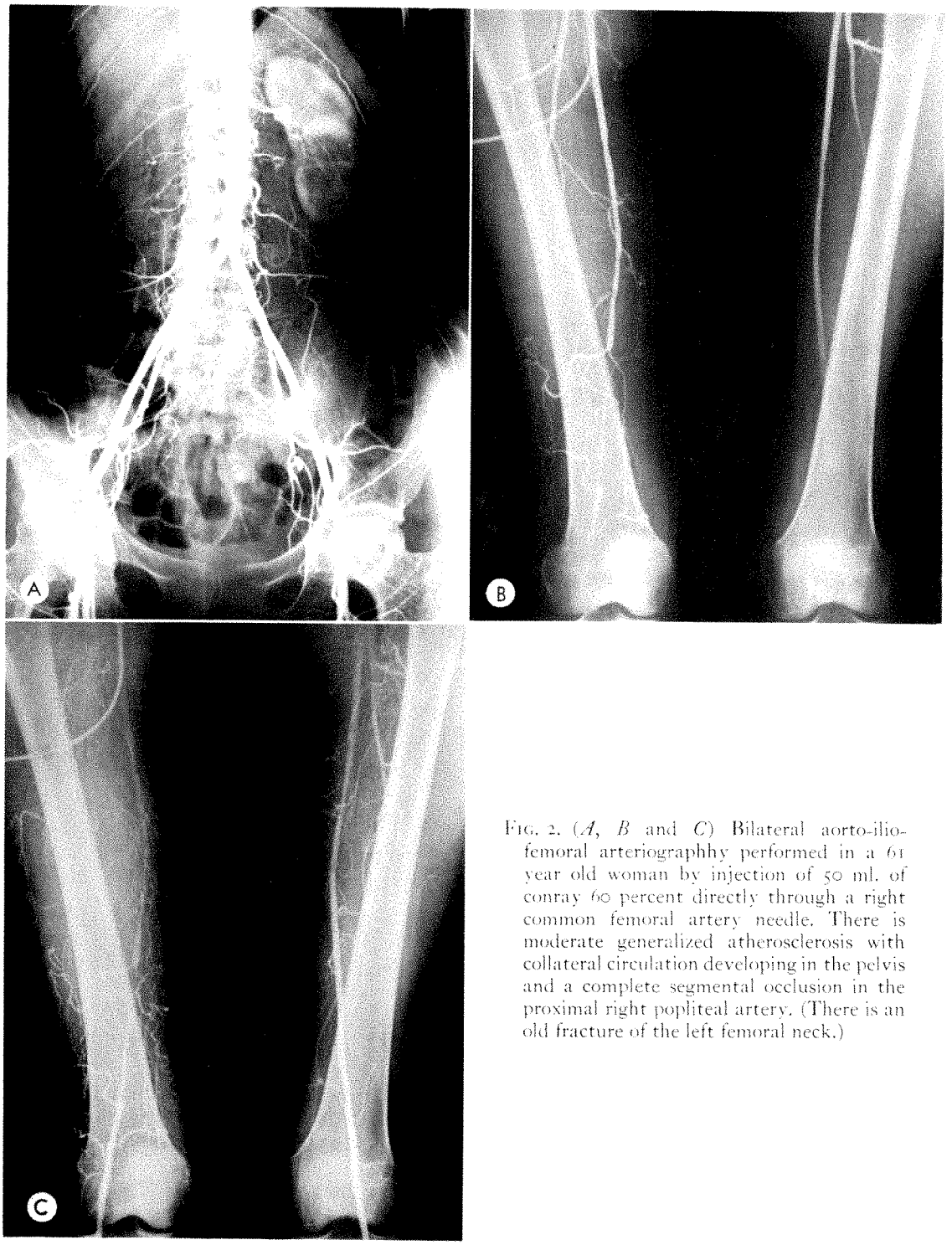


Fig. 2. (*A*, *B* and *C*) Bilateral aorto-ilio-femoral arteriographhy performed in a 61 year old woman by injection of 50 ml. of conray 60 percent directly through a right common femoral artery needle. There is moderate generalized atherosclerosis with collateral circulation developing in the pelvis and a complete segmental occlusion in the proximal right popliteal artery. (There is an old fracture of the left femoral neck.)

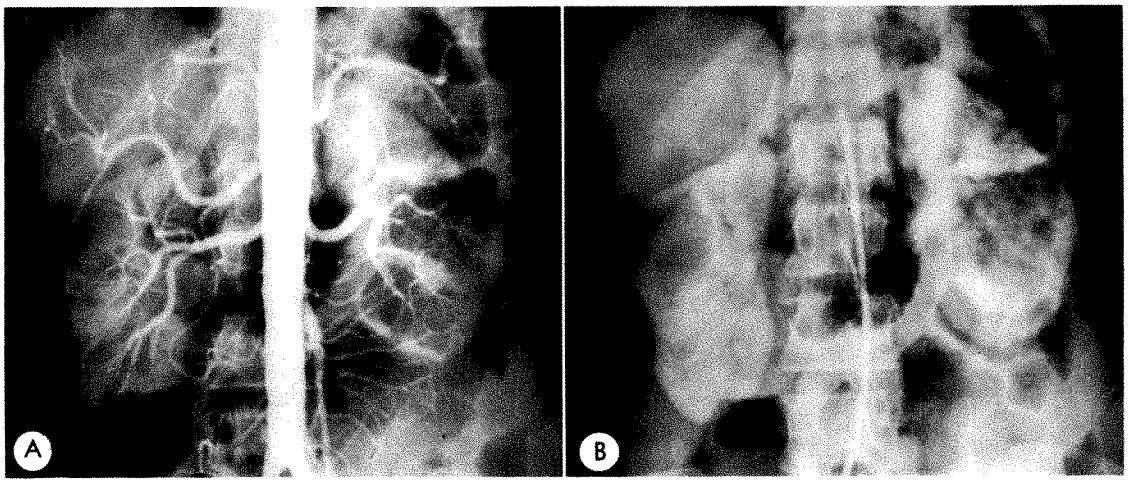


Fig. 3. (*A* and *B*) Retrograde catheter abdominal aortogram of a 45 year old woman by injection of 45 ml. of 60 per cent conray through a yellow Ödman-Ledin catheter introduced percutaneously from the right common femoral artery. There is a benign cortical cyst in the right kidney, displacing smaller intrarenal arteries and producing a characteristic radiolucent filling defect.

successfully completed. In 3 other patients extravasation of contrast material occurred at the site of femoral artery or vein puncture. Although 2 of these patients (1 conray, 1 hypaque) tolerated the incident quite well, 1 patient (conray) became hypotensive. He responded quickly to 0.5 ml. of intravenous methoxamine hydrochloride. The examinations were successfully completed in all 3 patients after the needles were repositioned. One patient receiving conray during retrograde catheter abdominal aortography developed on the side of the femoral artery puncture an absent posterior tibial pulse and a cyanotic great toe which reverted to normal within 24 hours on anticoagulant therapy. This was probably due to the loosening of an atherosclerotic plaque or thrombus with embolization to the lower extremity. Two patients developed shaking chills and headache, 1 following catheter aortography and 1 after pelvic phlebography and inferior vena cavagraphy. These proved to be sterile pyrogenic reactions traced to 2 bottles of contaminated saline. Several patients developed clinically insignificant hematomas at the site of arterial puncture, quickly controlled by further manual compression.

DISCUSSION

The physical properties of conray 60 per cent compare satisfactorily with other intravascular contrast agents used for the same purposes. It is a clear, colorless liquid with a high iodine content and radiodensity. Conray is highly soluble at room temperatures, making it unnecessary to warm it prior to injection. Its low viscosity provides the advantages of ease and speed of injection and permits the rapid delivery of large volumes for angiography.

Further advantages of conray 60 per cent are its low protein-binding characteristics and the fact that upon incubation with blood it does not cause red blood cell crenation seen with acetrizoate or rouleaux formation seen with N-methylglucamine diatrizoate.[1] Finally, animal experiments in the laboratory indicate that the toxicity of conray is extremely low.[3,5]

For peripheral angiography there is essentially no difference in patient tolerance to conray, renografin, and hypaque in the concentrations and volumes used in this study. Conray is as free from significant side effects and is at least as well tolerated as similar doses of intra-arterial renografin or intravenous hypaque. Furthermore,

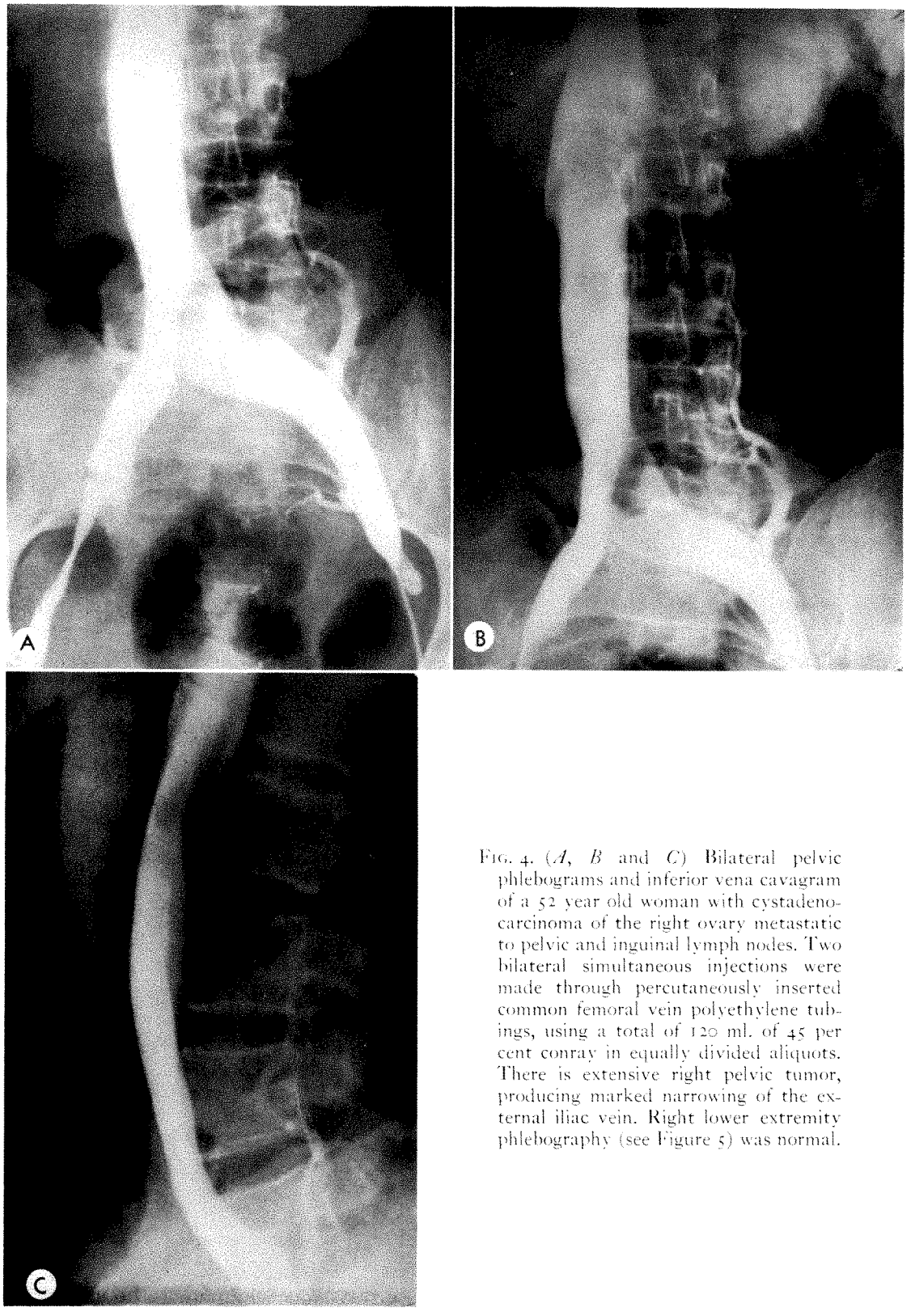


FIG. 4. (*A*, *B* and *C*) Bilateral pelvic phlebograms and inferior vena cavagram of a 52 year old woman with cystadenocarcinoma of the right ovary metastatic to pelvic and inguinal lymph nodes. Two bilateral simultaneous injections were made through percutaneously inserted common femoral vein polyethylene tubings, using a total of 120 ml. of 45 per cent conray in equally divided aliquots. There is extensive right pelvic tumor, producing marked narrowing of the external iliac vein. Right lower extremity phlebography (see Figure 5) was normal.



Fig. 5 (*A*, *B* and *C*) Normal right lower extremity phlebograms in the same patient shown in Figure 4, performed by injecting 45 ml. of 60 per cent conray in a superficial vein over the dorsum of the foot.

there were no severe reactions in 341 injections of conray which could be attributed to the drug.

A greater percentage of patients complained of generalized warmth following intravenous than intra-arterial injections.

Table VI

TECHNICAL COMPLICATIONS IN 502 EXAMINATIONS*

	Femoral Arteriography	Aorto-Ilio-Femoral Arteriography	Catheter Aortography	Pelvic and Inferior Vena Cavagraphy
Hypotension	1 (C)	1 (C)	1 (R)	—
Extravasation	1 (C)	1 (C)	—	1 (H)
Embolus	—	—	1 (C)	—
Pyogenic reaction	—	—	1 (R)	1 (C)

* No technical complications were encountered in performing lower or upper extremity phlebography or superior vena cavagraphy. The type of contrast material used is indicated in parenthesis.

On the other hand, a far greater percentage of patients complained of pain following intra-arterial than intravenous injections. The degree of pain is a subjective response related to the patient's pain threshold and the nature and amount of premedication given and, therefore, is difficult to evaluate in individuals. It often appears to be directly proportional to the speed and concentration of contrast material reaching smaller peripheral arteries and inversely proportional to the extent of development of collateral circulation. There was no appreciable difference among the 3 contrast agents tested in the amount of pain induced by the injection. No matter how severe nausea and vomiting was following an injection of contrast material, patients did not become nauseated or vomit following subsequent injections during the same examination.

No attempt was made to relate roentgenographic quality of the examination to the type of contrast material used. It is impossible to make this judgment, because it depends upon a great variety of factors unrelated to the contrast agent, such as centimeter thickness of the part roentgenographed, technical factors (kilovoltage, milliamperage, time), type of generator, screens, film speed, focal-film distance, patient cooperation, etc.

SUMMARY

A double blind clinical comparison has been made of conray 60 per cent with renografin 60 per cent in peripheral arteriography and with hypaque 50 per cent in peripheral phlebography. A total of 502 examinations and 755 injections were performed in 460 patients. The physical properties of conray compare satisfactorily with renografin and hypaque, and essentially no difference in patient tolerance to these contrast agents was found in the clinical studies. It is concluded that conray is a safe, effective intravascular contrast agent, well-tolerated by patients and suitable, even in large volumes, for peripheral angiography.

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The invaluable assistance of Esther Rowell, R. N., is gratefully acknowledged.

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* Deceased.

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Forty-seventh Annual Meeting: Roosevelt Hotel, New Orleans, La., April 8–10, 1965.

EDITORIAL

THE FORTY-SIXTH ANNUAL MEETING OF THE AMERICAN RADIUM SOCIETY

THE American Radium Society held its Forty-sixth Annual Meeting in White Sulphur Springs at The Greenbrier in the beautiful setting of the West Virginia upland Allegheny valley. The luxurious accommodations in a superbly managed hotel of incomparable decor offered every kind of sport, a cuisine by expert culinarians and a variety of entertainment. Attendance by members and guests was again at a high level with a total registration of 402, representing 119 members, 137 scientific guests and 146 ladies.

The meeting was officially opened on Monday morning, April 13 in the hotel theatre. Dr. Charles L. Goodhand, President of the West Virginia State Medical Association, welcomed the Society to White Sulphur Springs and stated that the Society represented an organization which coordinated research and teaching with the best in the practice of medicine. Dr. Charles G. Stetson of New York delivered the Presidential Address, his title being "Cancer Management and Radiotherapy Centers." He pointed out that approximately 40 per cent of new cancer patients are treated in hospitals of less than 150 beds. Many of these patients with certain types of cancer receive excellent care, but for others there is a lack of availability of highly skilled cancer surgeons, pathologists, radiotherapists and megavoltage radiation equipment. Many more cancer centers need to be established on a voluntary basis in our urban centers. There is also a need for an open-staff policy for qualified professional personnel at hospitals which have developed specialized facilities. Dr. Stetson quoted several of the recommendations of the British Ministry of Health on the Organization of Radiotherapy Services for the Treatment of Cancer: (1) Radiotherapy should be under the control of radiotherapists engaged solely in that work; (2) the center should be a department of a large hospital; (3) megavoltage therapy is essential for the treatment of cancer; (4) regional centers should be linked with a university in the area; (5) associated treatment centers should be established; (6) peripheral clinics are required for preliminary diagnosis, consultation on treatment and follow-up examination; and (7) radiotherapy beds should be provided in adequate numbers. He mentioned the Radiation Center of Fort Worth, Texas, as one of the finest examples of a cooperative municipal effort. The small community hospital may associate itself with an urban cancer facility and serve as a cancer detection center. An example of this effort is the Green Point City Hospital of Brooklyn, which is operated by the Mount Sinai Hospital in New York City, 15 miles away. Patients requiring radiotherapy are referred to the Radiotherapy Center at Mount Sinai Hospital. Dr. Stetson decried the short, one-year training period in cancer management accepted by specialty boards for certification in otolaryngology, obstetrics and gynecology, and radiology.

The first scientific session under the chairmanship of Dr. Juan A. del Regato consisted of presentations on the comparative place of irradiation and surgery in the treatment of bronchogenic carcinoma, radiographic and pathologic correlation as a guide to operability in post-irradiated lung cancer patients, results of radiotherapy in patients with inoperable carcinoma of the lung whose status was established at exploratory thoracotomy, and radiation injury of the lung.

The second part of the Monday morning session included a variety of subjects. The first 3 papers dealt with the potentialities for fast neutron beam radiotherapy, a compact radium safe employing gas sterilization and mammography as an aid in detection of cancer of the breast.

Dr. John S. Laughlin served as chairman of a symposium on Cancer Detection by Radioisotope Scanning. Dr. John P. Storaasli of Cleveland, presented a new approach on the detection of neoplastic lesions in organs and evaluation of an improved scintiscanner. Dr. Edward B. Schlesinger of New York Neurological Institute presented isotope techniques in the diagnosis of brain tumors and Dr. Ernest Greenberg of the New York Memorial Hospital spoke on scanning techniques in the detection of bone lesions using Ca^{47} and Sr^{85}. Dr. Merrill A. Bender of Roswell Park Memorial Institute, Buffalo, New York, spoke on tumor detection and the visualization of dynamic processes with the autofluoroscope, an ingenious piece of apparatus containing 300 detector crystals.

Dr. John L. Pool was chairman of the second scientific session during the first half of the Tuesday morning program. Dr. Gordon McNeer of New York, presented a comprehensive discussion on the life history of malignant melanoma from experience gained over many years at the Memorial Hospital for Cancer and Allied Diseases. Dr. Magnus I. Smedal spoke on rectosigmoid carcinoma treated by 2 mev. roentgen rays and demonstrated a bowel protecting technique. Dr. Giulio J. D'Angio emphasized the importance of emergency treatment for the superior mediastinal syndrome in children with cancer. Dr. Martin Van Herik of The Mayo Clinic reported on fever as a complication of radiation therapy for carcinoma of cervix. Dr. Felix Rutledge of Houston, spoke on the selected use of radiation in pelvic lymphadenectomy in the treatment of carcinoma of cervix.

In the latter half of the Tuesday morning session, Dr. Harald H. Rossi gave an interesting discussion on the biophysical considerations for the existence of a threshold for radiation effects. He stated that the conclusions reached should be of significance in radiation protection, radiobiology and radiotherapy.

A symposium on the palliation of advanced cancer was held under the chairmanship of Dr. Arthur I. Holleb. Dr. Joseph F. Farrow spoke on general surgical methods, Dr. Vernon H. Mark on neurosurgical methods, including stereotactic thalamotomy for the relief of pain, Dr. Rita G. Jacobs on anesthesia techniques, Dr. Raymond W. Houde on control of pain by analgesics, Dr. Juan A. del Regato on radiotherapy methods, Dr. Michael J. Brennan on hormonal and nonhormonal chemotherapy and Dr. Oscar L. Morphis on the use of hypnosis. This symposium covered a large area in the treatment of cancer and considerable discussion was elicited from the audience.

The highlight of the meeting was the presentation of the Annual Janeway Lecture. Dr. Edith H. Quimby introduced Dr. Harold W. Dargeon as an experienced Pediatric Oncologist with a background of 40 years in the field of Pediatrics. He has been Director of the Pediatric Service at Memorial Hospital for many years and is Emeritus Clinical Professor of Pediatrics at Cornell Medical School. Dr. Dargeon spoke on the treatment of reticuloendotheliosis and reviewed 62 cases which included patients with solitary eosinophilic granuloma, disseminated Hand-Schüller-Christian syndrome and malignant Letterer-Siwe's disease. The course of the disease and treatment were outlined in a general way. The latter included biopsy, chemotherapy, radiation therapy and surgery for complications.

In the evening, a reception for members and guests of the Society was held in the beautiful ballroom of the hotel and sponsored by the Radium Chemical Company of New York.

The third scientific session on Wednesday morning was under the chairmanship of Dr. Justin J. Stein. The early part of this meeting included subjects related to

the management of cancer of the head and neck and of the esophagus. Dr. Rodney R. Million presented a paper on malignant epithelial tumors of the nasopharynx and was followed by Dr. Charles L. Martin on the treatment of epithelioma of the oropharynx. Other subjects discussed included combined interstitial and external therapy in the treatment of head and neck tumors, management of residual or recurrent cancer following radiation therapy for squamous cell carcinoma of the oropharynx and preoperative irradiation for cancer of the esophagus.

Following considerable discussion on the papers presented in the early morning session, the main guest speaker from abroad, Dr. Frank Ellis, radiotherapist from Churchill Hospital of Oxford, England, spoke on rational collaboration between surgeon and radiotherapist. Dr. Ellis pointed out the importance of coordination of radiotherapy and the various disciplines of medicine, especially related to the rationale of combining irradiation and surgery in the treatment of cancer. He showed many instances of the value of preoperative and postoperative radiation therapy. He emphasized the technique of postoperative therapy with radium needles, principally by the after-loading technique using polythene tubes. Many neoplasms which could not be cured by either method alone showed surprisingly good results by the combination of surgical treatment and radiotherapy.

The latter part of the Wednesday morning session was occupied by a workshop on statistics under the chairmanship of Dr. Antolin Raventos of the Hospital of the University of Pennsylvania. Dr. Howard B. Latourette of Iowa City spoke on the value and use of tumor registry data, Dr. Sidney J. Cutler answered the question on the presentation of the end results of cancer therapy, Dr. Marvin A. Schneiderman explained the pitfalls of using retrospective data in comparison treatments and Dr. James J. Nickson discussed the place of clinical trial in the future of radiotherapy. This symposium was followed by a question period and considerable discussion.

In the early evening, a reception was held in the ballroom which was later followed by The Annual Banquet held in the beautiful recently built Chesapeake Room. Dr. Charles G. Stetson introduced the members and wives at the speaker's table and with appropriate ceremony presented the Janeway Medal to Dr. Harold W. Dargeon for his outstanding achievements in the field of Pediatric Oncology. Members and guests were entertained by a talented dancing team and also participated in the dancing during the evening.

The final and fourth scientific session under the chairmanship of Dr. Milton Friedman encompassed papers on chemotherapy of metastatic testis cancer, the treatment of inoperable liver cancer by the intra-arterial infusion of chemotherapeutic and radioisotopic agents, clinical therapeutic (radioactive) lymphangiography, current status of regional oxygenation and radiation and treatment of carcinoma of the bladder by supervoltage linear accelerator radiotherapy.

The second half of the last scientific session included presentations on iridium 192 as a radium substitute, evaluation of tolerance and effectiveness of the cobalt 60 moving strip, experience with high energy (35 mev.) electron beam therapy, diagnosis and treatment of malignant synovioma, radiation hepatitis, experience with 883 patients with malignant lymphoma and the effect of radiation therapy on the survival time of women with recurrent mammary carcinoma.

The Forty-sixth Annual Meeting of the American Radium Society again proved to be successful by its large attendance, by the high quality of its scientific program and by the attraction of gracious living in White Sulphur Springs. The members and guests of the Society owe a debt of gratitude to its President, Dr. Charles G. Stetson, to Dr. Henri LeClair and the members of his Arrangements Committee and to the management of The Greenbrier for its efforts in prividing many conveniences and recreational facilities.

Harry Hauser, M D

NEWS ITEMS

AMERICAN RADIUM SOCIETY ELECTS NEW OFFICERS

At the Forty-sixth Annual Meeting of the American Radium Society held at the Greenbrier Hotel, White Sulphur Springs, West Virginia, April 13–16, 1964, the following officers were elected: *President:* Joseph H. Farrow, New York, N. Y.; *President-Elect*: Justin J. Stein, Los Angeles, Calif.; *1st Vice-President:* Robert C. Hickey, Madison, Wisc.; *2nd Vice-President:* Marvin Williams, Rochester, Minn.; *Secretary:* John L. Pool, New York, N. Y.; *Treasurer:* Milton Friedman, 1016 Fifth Ave., New York 28, N. Y.

The Forty-seventh Annual Meeting of the Society will be held at the Roosevelt Hotel, New Orleans, La., April 8–10, 1965.

DR. L. HENRY GARLAND HONORED

The radiologists of his own state recently honored Dr. L. Henry Garland on the completion of 30 years as Honorary Secretary of the State Radiological Society. At the Annual Meeting of the Society held in Los Angeles on March 22, 1964, Dr. Lowell S. Goin, on behalf of the officers and members, presented him with an engraved silver and oak plaque in appreciation of his long and devoted service.

THE CANADIAN ASSOCIATION OF RADIOLOGISTS

At the 27th Annual Meeting of the Canadian Association of Radiologists held at Vancouver, B. C., March 3, 1964, the following officers were elected for the year 1964: *President:* Ross A. Lobb, M.D.; *Vice-President:* Henri Lapointe, M.D.; *Honorary Secretary-Treasurer:* D. J. Sieniewicz, M.D.; *Associate Honorary Secretary-Treasurer:* M. Dufresne, M.D.

SOUTHERN RADIOLOGICAL CONFERENCE

At the Eighth Annual Meeting of the Southern Radiological Conference held at the Grand Hotel, Point Clear, Alabama, January 31, and February 1 and 2, 1964, the following officers were elected: *Chairman:* Dr. Monte A. Lauter, Mobile, Alabama; *Vice-Chairman:* Dr. Seymour Fiske Ochsner, New Orleans, Louisiana; *Secretary-Treasurer:* Dr. Marshall Eskridge, Mobile, Alabama (re-elected).

The Ninth Annual Meeting will be held at the Grand Hotel, Point Clear, Alabama, January 29, 30 and 31, 1965.

X-RAY EXPOSURE STUDY

The U. S. Public Health Service began on April 1, 1964, the second of a series of national x-ray studies designed to provide additional information about the x-ray experience of the U. S. population, as announced by Surgeon General Luther L. Terry.

These studies are being conducted jointly by the Division of Radiological Health and the National Health Survey of the Public Health Service. A report on the first study covering x-ray visits from July 1960–June 1961 was published by the National Health Survey in October, 1962.

The study was planned by an advisory group headed by Richard H. Chamberlain of Philadelphia, and including ACR members, Russell H. Morgan of Baltimore, Harold O. Wyckoff of Washington, E. Dale Trout of Corvallis, Oregon, Carl Braestrup of New York City, and Robert O. Gordon of Philadelphia, among others.

A number of efforts have been made in the past to obtain information about x-ray exposure in this country. These have been limited to selected populations or to certain types of examinations. The current study will be comparable in scope to the comprehensive studies made in other countries as, for instance, the "Adrian study" made in Great Britain during the 1950's.

The current study planners had available a report on a field trial conducted in Berks County, Pennsylvania, during the summer

of 1963 to test the methods and procedures to be used in the national study. A vital factor in the success of the field trial was the excellent cooperation of physicians, dentists and other practitioners who were asked to participate. A summary of the feasibility report appeared in the March issue of *Public Health Reports* (Gitlin, J. N. *et al.*, 1964, *79*, 185–192).

The national study will extend the information published in the National Health Survey Report by providing radiographic, fluoroscopic and therapeutic exposure estimates for a representative sample of the U. S. population. A third study is planned in which these exposure data will be translated into dose estimates to specific body organs. This will be accomplished through continued collaboration with radiologists and physicists engaged in dosimetric research.

The data collection phase of the current study which began April 1, 1964, consists of household interviews with the purpose of obtaining x-ray visit information for the preceding 3 month period. The survey will cover approximately 10,000 households and 32,000 persons. From May through September, 1964 follow-up questionnaires and film packs, designed to provide the required exposure information, will be mailed to practitioners or facilities identified in the household interview survey. It is estimated that approximately 6,000 x-ray facilities will be involved during this period. In each case, authorization to provide the requested information will have been obtained from the household respondent. The cooperation of all practitioners or facilities receiving follow-up requests is essential to meet the objectives of the study.

All information pertaining to individual patients, practitioners and facilities will be treated confidentially; only statistical summaries will be made available for publication.

For further information write to Dr. Donald R. Chadwick, Chief, Division of Radiological Health, U. S. Public Health Service, Washington, D. C. 20201.

NATIONAL BUREAU OF STANDARDS

Safe Handling of Radioactive Materials. Handbook 92, March 9, 1964, 107 pages, 40 cents.

This Handbook was prepared by the National Committee on Radiation and Measurements and is designed to help users to handle radionuclides without exposing themselves or others to doses in excess of established safety limits. The book covers control of radiation exposure; instruction and supervision of personnel; physical safeguards; procedural safeguards; radiation instrumentation, such as personnel meters, fixed monitors, sample counters, and so on; transportation of radioactive materials; and disposal of radioactive waste.

Handbook 92 supersedes NBS Handbook 42, *Safe Handling of Radioactive Isotopes*. In the interval since Handbook 42 was published in 1949, radionuclides have come into much wider use in research, medicine, industry, and agriculture. Their applications in radiography, level and thickness gaging, food processing, well logging, and radiotracer testing have introduced problems of radiation safety in many fields, but this diversified use has also broadened the base of experience in their safe handling.

The principles of radiation protection, first gathered together in Handbook 42, have been expanded and kept up to date in more recent publications, which, for the assistance of the reader, are listed in the present Handbook. Also referenced are other Handbooks where more detailed information can be obtained, such as accurate determinations of radiation doses and other complex technical problems outside the scope of Handbook 92.

Shielding against Gamma Rays, Neutrons and Electrons from Nuclear Weapons. A review and bibliography by J. H. Hubbel and L. V. Spencer. Monograph 69, Feb. 24, 1964, 36 pages, 30 cents.

This Monograph discusses the problem of predicting dose rates and of estimating the effectiveness of shielding from radiations resulting from nuclear explosions. A number of existing calculations and supporting experiments regarding the penetration and diffusion of gamma rays, neutrons, and electrons through air and bulk materials are summarized.

The problem of shielding against radiation due to nuclear weapons involves estimation of the radiation dose in an arbitrary configuration of radiation sources and shielding materials. In making such estimates, data for simple structure types have been very useful. This is because a complicated structure can often be schematized as a combination of simple structures. The infinite, homogeneous medium, *i.e.*, the total lack of structure, may be considered the simplest case, as well as the most useful.

The Monograph is primarily a catalog and bibliography of experiments and calculations relating to simple configurations, including the infinite, homogeneous medium. It extends the documentation of *Structure Shielding against Fallout Radiation from Nuclear Weapons* (NBS Monograph 42), and indicates the availability of additional data, calculations, and corroborative experiments.

A selection of 485 references from the unclassified literature is given, of which 388 are cataloged. These cataloged references include radiation field studies ranging from the point-source, infinite-medium situation up through such complicated geometries as foxholes, shelters, and conventional structures.

Orders for these 2 publications can be placed with the Superintendent of Documents, U. S. Government Printing Office, Washington, D. C. 20402. Foreign remittances must be in U. S. exchange and should include an additional one-fourth of the publication price to cover mailing costs.

BOOK REVIEWS

Books sent for review are acknowledged under: Books Received. This must be regarded as a sufficient return for the courtesy of the sender. Selections will be made for review in the interest of our readers as space permits.

BONE CHANGES IN HEMATOLOGIC DISORDERS; (ROENTGEN ASPECTS): A MOUNT SINAI HOSPITAL MONOGRAPH. By John E. Moseley, M.D., Associate Attending Radiologist, The Mount Sinai Hospital, New York; Director, Department of Radiology, Sydenham Hospital, New York. Cloth. Pp. 261, with many illustrations. Price, $9.50. Grune & Stratton, Inc., 381 Park Avenue S., New York 16, N. Y., 1963.

This monograph consists basically of a series of articles on the roentgen aspects of bone changes in hematologic disorders published in the *Journal of the Mt. Sinai Hospital* between 1959 and 1962. To this has been added a discussion of the bone changes in osteopetrosis and a short concluding commentary.

Because the application of physicochemical, genetic, immunologic and anthropologic techniques to the study of hematologic disorders has led to some stunning revelations regarding the basic nature of these conditions, and, at the same time, some refinements in the roentgen diagnosis of the skeletal manifestations of these disorders have taken place, the author considered it worthwhile to condense our current knowledge regarding the bone changes in hematologic disorders into one central source. This body of information is complemented by his personal observations and some of the lingering misconceptions are pointed out.

Utilizing the roentgenographic appearance of the skeletal changes as the central theme, the author presents a beautifully correlated clinical, pathologic and roentgenologic discussion of the anemias, classic hemophilia and Christmas disease, leukemia and myelosclerosis, osteopetrosis, multiple myeloma, the reticuloendothelioses and the malignant lymphomas.

While this monograph, with its remarkably detailed illustration of roentgen pathology, is primarily addressed to radiologists, its Atlas type quality will also be of considerable aid to the hematologist, pathologist, orthopedic surgeon, pediatrician, and internist.

A thorough cross index and a comprehensive bibliography at the end of each section add to the practicalness and reference value of this unreservedly recommended little classic on hematoskeletal disorders.

DONALD M. MONSON, M.D.

LA GRANULOCITOPOIESI: FISIOPATOLOGIA E CLINICA. Collana di Monografie a cura della Fondazione Gonassini. By Carlo Sacchetti, dell'Istituto di Patologia Speciale Medica della'Università di Genova, Genova, Italy. Cloth. Pp. 191, with 29 illustrations and 26 tables. Price, L. 3,500. L. Capelli, Via Farini 6, Bologna, 1962.

Among the blood cells, the neutrophilic granulocytes are those most frequently involved in a great variety of pathologic conditions both clinical and subclinical—from true hemopathy to infectious diseases, provided that they arise as a consequence of particular therapies or because of occasional exposure to harmful agents.

Up to the present, the difficulties encountered in the study of granulocytes and granulocytopoiesis have restricted the clinical evaluation of the degree of leukocytosis and leukopenia to the blood count and the morphologic examination of the bone marrow—undoubtedly useful factors but too generic and consequently of limited importance. In order to obtain a more complete picture, it was essential to be in possession of additional facts concerning the renewal of the granulocytes.

The introduction of particular techniques for staining human granulocytes has made possible their study in accordance with exact quantitative and dynamic standards. The most important material from which the investigations illustrated in the monograph are developed are related to the following 3 parameters: (1) In the blood, the granulocytes are divided into 2 parts of almost equal number and are interchangeable among themselves; (2) the average life of blood granulocytes is very brief (about 7 hours in normal conditions); approximately 48 hours are required for all the granulocytes present in the blood to be replaced by new cells

arising from the bone marrow; (3) there is a reserve of fully developed granulocytes in the bone marrow ready to pass into the blood stream; this reserve is very large (about ten times the bulk of the blood granulocytes) and is a functional element of particular importance; it can alone ensure the normal replenishment of cells in the blood (the count therefore remaining unaltered) for 6–7 days, even if the reproduction of bone marrow is suspended.

Radioisotope techniques have paved the way to efficacious use of simple tests such as Arneth's formula and mobilization of the reserve in order to obtain an accurate prognosis; the application of these methods of research, therefore, extends beyond the strictly clinical field.

The monograph is divided into 3 parts: the first is devoted to the physiopathology of granulopoiesis; the second deals with the clinical study of granulopoiesis; and the third covers an exposition of the techniques and criteria of interpretation and calculation. Accompanying this exposition are 26 tables and 29 illustrations.

An analytic index, as well as an index of the illustrations and tables, facilitates consultation.

EMILIO GANASSINI

BOOKS RECEIVED

HIGH SENSITIVITY COUNTING TECHNIQUES. International Series of Monographs on Electronics and Instrumentation. Volume 20. By D. E. Watt, United Kingdom Atomic Energy Authority, Chapelcross, Annan, Scotland; and D. Ramsden, United Kingdom Atomic Energy Authority, Winfrith, Dorset, England. Cloth. Pp. 348, with many illustrations. Price, $12.00. A Pergamon Press Book, distributed by The Macmillan Company, 60 Fifth Avenue, New York 11, N. Y., 1964.

EFFECTS OF IONIZING RADIATION ON THE REPRODUCTIVE SYSTEM. Proceedings of an International Symposium held at Colorado State University, Fort Collins, Colorado. Edited by William D. Carlson, Department of Veterinary Medicine, Colorado State University; and F. X. Gassner, late of the Department of Veterinary Medicine, Colorado State University. Cloth. Pp. 478, with many illustrations. Price, $14.00. A Pergamon Press Book, distributed by The Macmillan Company, 60 Fifth Avenue, New York 11, N. Y., 1964.

THE LUNG AND ITS DISORDERS IN THE NEWBORN INFANT. Volume I in the Series, Major Problems in Clinical Pediatrics, Alexander J. Schaffer, Consulting Editor. By Mary Ellen Avery, A.B., M.D., Assistant Professor of Pediatrics, Johns Hopkins University School of Medicine; and Pediatrician-in-charge, Newborn Nurseries, The Johns Hopkins Hospital. Cloth. Pp. 224, with many illustrations. Price, $7.50. W. B. Saunders Company, Philadelphia, Pa., 1964.

THE RADIOLOGIC CLINICS OF NORTH AMERICA. SYMPOSIUM ON RADIOLOGIC DIAGNOSIS OF ACUTE ABDOMINAL DISEASE. Volume II. No. 1. Guest Editor, Harry Z. Mellins, M.D. Cloth. Pp. 188, with many illustrations. W. B. Saunders Company, Philadelphia, Pa., 1964.

ATLAS OF TOPOGRAPHICAL AND APPLIED HUMAN ANATOMY. Volume II. Thorax, Abdomen and Extremities. By Eduard Perndopf. Edited by Dr. Helmut Ferner, Professor and Director of the Anatomical Institute, University of Heidelberg, Germany. Translated from German by Dr. Harry Monsen, Associate Professor of Anatomy, Department of Anatomy, University of Illinois College of Medicine, Chicago, Ill. Cloth. Pp. 421, with 378 illustrations. Price, $37.50. W. B. Saunders Company, Philadelphia, Pa., 1964.

DIE MENISCUSLAESION: PRAKTISCHE PROBLEME DER KLINIK, ARTHROGRAPHIE UND THERAPIE. By Priv.-Doz. Dr. P. Ricklin, Männedorf bei Zürich; Priv.-Doz. Dr. A. Rüttimann, Zürich; and Priv.-Doz. Dr. M. S. Del Buono, Zürich. Cloth. Pp. 156, with 219 illustrations. Price, Ganzleinen DM 48.-. Georg Thieme Verlag, Stuttgart 1, Germany. In the U.S.A. and Canada, Intercontinental Medical Book Corporation, New York 16, N.Y., 1964.

PNEUMOGINECOGRAFIA E LAPAROSCOPIA: UTILITÀ DELLA LORO ASSOCIAZIONE NELLA DIAGNOSI DELL' PELVIPATIE. By Prof. Luigi Tenti, Istituto di Radiologia, Ospedale di Varese; Dott. Italo Belli, Istituto di Radiologia, Ospedale di Varese; and Dott. Gianfranco Nocca, Servizio di Laparoscopia, Ospedale di Varese. Cloth. Pp. 168, with many illustrations. Edizioni Minerva Medica, Torino, Italy, 1963.

OAK RIDGE INSTITUTE OF NUCLEAR STUDIES. Medical Division Research Report for 1963. Operating under contract AT-40-1-Gen-33 with the United States Atomic Energy Commission. Paper. Pp. 105, with some illustrations and graphs. Price $2.25. Office of Technical Services, Department of Commerce, Washington 25, D. C.

RADIOLOGY: CUMULATIVE INDEX (V). Volumes 70–79, 1958–1962. Foreword by Howard P. Doub, M.D., Editor. Cloth. Pp. 399. Price, $16.50. Radiological Society of North America, Inc., 713 East Genesee Street, Syracuse, N. Y., 1964.

ABSTRACTS OF RADIOLOGICAL LITERATURE

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ROENTGEN DIAGNOSIS

Head

Morton, W. Ross, and Turnbull, William. Blowout fractures of the floor of the orbit. *Canad. M. A. J.*, Jan 11, 1964, *90*, 58–61. (From: Royal Victoria Hospital, Montreal, Quebec, Canada.)

The term, "blowout fracture of the floor of the orbit," as used originally by Converse and Smith signifies a fracture of the floor of the orbit with herniation of orbital contents into the maxillary sinus. This is not easily diagnosed and since the authors saw a series of 5 patients with such an injury during a 2 month period, it seemed advisable to report them.

The floor is said to be the weakest portion of the orbit and a blunt blow directed at the contents of the orbit tends to fracture the weakest area, and forces the contents through into the maxillary sinus. Since the roentgenologic findings may simulate sinusitis, mucocele, etc., the authors urge the use of laminagraphy in the Waters' projection. Surgical correction is through the lower lid and aims at repairing the defect with teflon or some type of mesh.—*Richard E. Kinzer, M.D.*

Dooley, Donald M., and Perlmutter, Irwin. Spontaneous intracranial hematomas in patients receiving anticoagulation therapy: surgical treatment. *J.A.M.A.*, Feb. 8, 1964, *187*, 396–398. (Address: Dr. Dooley, 4685 Ponce de Leon Boulevard, Coral Gables, Fla.)

Intracranial hematomas may be caused by trauma, hypertension, aneurysms, arteriovenous malformations, and angiomas. In addition to these causes the authors report 3 cases of hematomas arising spontaneously in patients being treated with anticoagulants.

Two of the 3 patients had been on prolonged therapy with warfarin, the other only for a matter of weeks. All 3 had been in the "therapeutic range" with a prothrombin time of 25–30 seconds when apparently spontaneous intracranial bleeding occurred. Two of the hematomas were intracerebral, and the other was subdural. Significant and progressive neurologic signs were present and following treatment of depressed clotting mechanism, cerebral arteriography was performed with localization of the hematomas. Surgical removal was followed by recovery in all 3 cases.

In an addendum the authors report a fourth case successfully operated following the preparation of this paper.—*Donald S. Linton, Jr., M.D.*

Neck and Chest

Brown, B. St. J., Dunbar, J. S., and MacEwan, D. W. Foreign bodies in the tracheobronchial tree in childhood. *J. Canad. A. Radiologists*, Dec., 1963, *15*, 158–171. (From: The Montreal Children's Hospital, Montreal, Quebec, Canada.)

Review of 160 patients admitted to Montreal Children's Hospital, with one or more foreign bodies in the tracheobronchial tree revealed that the commonest age group involved is from 1 to 3 years, accounting for 76 per cent of all patients. The majority was diagnosed within 2 months of aspiration, but in 5 patients the foreign bodies had certainly been present for over 1 year. The vast majority of foreign bodies could be classed as food, with peanuts (61.2 per cent) the most common offender. Timothy grass was notorious for remaining undetected for long periods of time. In 10 per cent of the patients the condition was unsuspected on admission to hospital.

The most frequent roentgenographic sign was obstructive overinflation of one or more lobes, which occurred in 68.1 per cent of patients. This was about five times more common than the next frequent finding—collapse of varying degree (13.8 per cent). Infiltration indicating some form of pneumonitis occurred as a presenting feature in 10.6 per cent of patients. The foreign body could be visualized (was opaque) in 9.4 per cent.

The foreign body lodged in the trachea in only 8.8 per cent of patients, but lodged in a main stem bronchus in 69.1 per cent, of which 40.5 per cent were in the right main stem bronchus and 26.4 per cent in the left. Those which passed farther distally usually lodged in a lower lobe bronchus, approximately 10 per cent on each side.

Pneumomediastinum occurred in 2 patients and pneumothorax in 3 patients. Lung abscess with extensive pneumonia was demonstrated in 1 patient. The most common complications were pneumonitis, which occurred in 5.6 per cent, and bronchiectasis in 3.1 per cent. The most significant factor in the development of complications appeared to be the duration before removal. In the 3 severe cases of bronchiectasis, many months passed before the foreign body was removed.

Two patients died, one a 20 month old boy who had aspirated 2 peanuts, obstructing all but the right upper lobe, and the other a 16 month old boy who died during bronchoscopy, possibly because of partial occlusion of the uninvolved left bronchus during repeated and only partially successful attempts to remove a peanut obstructing the right bronchus. The third patient developed anoxic brain damage. Initially this patient had been thought to have encephalitis and the presence of a foreign body in the tracheobronchial tree was overlooked.

Surgery in addition to bronchoscopy was necessary in 7 patients, including those requiring surgery for complications of foreign body aspiration.

There was agreement in roentgenologic and bronchoscopic findings in 84.4 per cent of cases. In the remaining 15.6 per cent the lack of correlation was variable in degree. In 3 cases the normally ventilating lung was thought to show collapse, but subsequent roentgenograms or review of roentgenograms revealed the true situation. In several patients the foreign bodies changed positions.—*Lois Cowan Collins, M.D.*

Marrocu, F., and Cossu, F. Tecnica personale per la visualizzazione del dotto toracico. (Personal technique for the visualization of the thoracic duct.) *Radiol. med.*, Nov., 1963, *49*, 945–953. (Address: Dott. Francesco Marrocu, Istituto Radiologia Medica Università, Cagliari, Italy.)

A new method of roentgenologic visualization of the thoracic duct is described.

The patient lies supine on the roentgenographic table and after the usual preparation a lymphatic vessel of the dorsal aspect of each foot is chosen for the injection of the contrast medium. About 15–25 cc. of lipiodol is injected in the two sides at the rate of 1 cc. every 6–7 minutes (in a few cases it might be necessary to inject up to 24–28 cc. in order to obtain a satisfactory opacification of the whole thoracic duct). The flow of the contrast medium along the lymphatic trunks is followed by frequent fluoroscopic observations. Once the arch of the thoracic duct, situated high in the cervical region, is opacified, the injection of the contrast medium is stopped, and the table is tilted almost vertically. In about 20 minutes the column of the contrast medium in the thoracic duct is about 10 cm. high. At this moment the patient is asked to inhale deeply a few times in order to force more of the contrast medium upward until in reaching the arch of the duct it passes into the venous current. Another useful aid is the Valsalva maneuver. A patient normally has to remain standing about 30 minutes and the duct appears opacified homogeneously in its entirety for 15–18 cm. The table is returned to the horizontal position when the duct is opacified in its entire course from its origin to its point of emptying in the venous current.

The roentgenograms are taken with the patient supine in the anteroposterior projection in which the arch is clearly demonstrated. The oblique views demonstrate the thoracic segment of the duct.

The demonstration of the thoracic duct is facilitated principally by the fact that the lipiodol is not soluble in the lymph and has a greater specific weight. Secondarily, due to the dynamics proper to the lymphatic trunks and the pressure with which the contrast medium is injected, a slow complete opacification of the duct is achieved.

At the end of the examination the patient is asked to remain in a sitting position for about 4 to 5 hours in order to avoid the possibility of the contrast medium passing into the venous current with a speed greater than normal.—*A. F. Goconi, M.D.*

Abdomen

Potvliege, R., Engelholm, L., Desneux, J., and Potvliege, P. Le nodule de Berg: confrontations cliniques, endoscopiques, radiologiques et anatomiques à propos de 3 ulcères présentant une microlacune centrale. (Berg's nodule: clinical, endoscopic, radiologic and anatomic comparative study of three cases of ulcer presenting a central microlacuna.) *J. belge de radiol.*, 1963, *46*, 346–357. (From: Hôpital Universitaire Brugmann, Bruxelles, Belgium.)

Three cases of ulcer with a very small depression at the center of the ulcer niche are presented. The analysis of these cases tends to confirm the opinion of Berg that the microlacuna at the center of the ulcer niche is due to a blood clot; hence the close relationship of this finding with the increased incidence of bleeding of such ulcer.

This roentgen sign should indicate a closer watch of the patient, but not necessarily surgical intervention.—*Jirair M. Sarian, M.D.*

Chodack, Paul, and Hurwitz, Alfred. Lymphangiectasis of stomach simulating polypoid neoplasm. *A.M.A. Arch. Int. Med.*, Feb., 1964, *113*, 225–229. (Address: Dr. Chodack, 1204 Ditmas Avenue, Brooklyn 18, N. Y.)

A proven case of lymphangiectasis of the stomach is described. According to the authors, only one case of probable lymphangiectasis of the stomach has been reported previously.

The authors' case was detected at roentgenologic examination and thought to be a polypoid carcinoma. At operation a cystic mass was found measuring $3\frac{1}{2}$ cm. in diameter. The mass and a portion of the mucous membrane were removed and the pathologic diagnosis was lymphangiectasis of the stomach.

It is suggested that compressible tumors of the stomach may be missed by the surgeon. Fewer will be overlooked if the surgeon uses the bimanual compression technique of palpation of the surgically exposed stomach.

Reproductions of roentgenograms and photomicrographs are included.—*John H. Harris, M.D.*

Moresi, Harry J., Jr., and Ochsner, Seymour Fiske. Megabulbus of the duodenum in the adult: report of a case associated with annular pancreas and ulceration. *Am. J.*

Digest. Dis., Feb., 1964, *9*, 170–174. (Address: 1514 Jefferson Highway, New Orleans 12, La.)

This report records the case of a 64 year old white male who was found to have a huge duodenal bulb, measuring approximately 11 cm. in diameter, a chronic penetrating postbulbar duodenal ulcer, and an annular pancreas above the ampulla of Vater.

Megabulbus of the duodenum is defined as localized enlargement of the duodenal bulb and is much rarer than megaduodenum involving the entire loop. Megabulbus results as a complication of postbulbar duodenal ulcer or an obstructing congenital band or annular pancreas. In the presence of an annular pancreas, duodenal ulcer may be found in as many as 30 per cent of the cases. When the duodenal loop is obstructed proximal to the ampulla of Vater, there is decreased alkaline backflow into the bulb favoring the development of ulceration.

The presence of a duodenal bulb in excess of 5 cm. in diameter usually signals the presence of underlying disease and diligent efforts must be made to demonstrate an ulcer of the mucosa of the bulb, a postbulbar ulcer, or an obstruction in the duodenum distal to the bulb. The presence of vigorous peristaltic activity in the bulb would suggest the presence of an obstructing lesion. Decreased peristalsis or atony of the duodenal loop would indicate the presence of a generalized or neurologic disorder producing a large bulb as part of megaduodenum.—*John H. Harris, M.D.*

VARADARAJAN, M. G. Amoeboma of the large intestine. *Punjab Med. J.*, Oct., 1962, *12*, 169–174. (Address: Radiologist, the Government General Hospital, Madras, India.)

The author reports a case of a patient who developed a mass in the right lower quadrant with symptoms of partial obstruction. He had a previous history of amebiasis. Barium studies showed a filling defect involving the cecum. Stool examination was negative for cysts or ova.

Because of the patient's history, a trial of antiamebic therapy was made. The condition improved markedly with subsequent disappearance of the mass.

The author points out that ameboma should be considered in a differential diagnosis of a mass of the cecum.—*George L. Sackett, Jr., M.D.*

FARMER, RICHARD G., HAWK, WILLIAM A., and TURNBULL, RUPERT B., JR. The spectrum of the Peutz-Jeghers syndrome: report of 3 cases. *Am. J. Digest. Dis.*, Nov., 1963, *8*, 953–961. (Address: Department of Gastroenterology, Cleveland Clinic, 2020 East 93rd Street, Cleveland 6, Ohio.)

The importance of recognizing the Peutz-Jeghers syndrome lies mainly in the malignant potential of the various polyposis syndromes. The major discrepancy of opinion regarding malignancy in the Peutz-Jeghers syndrome appears to be that the polyps are now believed to be hamartomas, which at times may be confused with malignancy.

The clinical hallmarks of the Peutz-Jeghers syndrome are mucocutaneous pigmentation and intestinal polyposis. The pigmentation is in the form of melanin deposits, almost invariably about the mouth, on the lips and the buccal mucosa and, less frequently, around the nose and eyes, and on the fingers and toes. In more than 90 per cent of such patients the polyps are found in the small bowel. They also occur throughout the gastrointestinal tract. Gastric and rectal polyps are relatively frequent. The syndrome is thought usually to be inherited as a simple mendelian dominant; sporadic cases have been considered to be a result of genetic mutation.

A father, daughter, and son each had the Peutz-Jeghers syndrome. The father had no abnormal pigmentation but had polyps both in the jejunum and in the colon, and had undergone a total colectomy and an ileostomy for a presumed diagnosis of familial multiple polyposis of the colon. The daughter had the complete syndrome, proved at laparotomy. The son had typical pigmentation, but no history or evidence of gastrointestinal polyps. Another brother, 24 years old, had no abnormal pigmentation and no roentgenographic evidence of polyps of the small bowel or the colon. The mother had no symptoms to suggest the presence of intestinal polyps.

The most frequent symptoms reported are abdominal pain due to intussusception by the small bowel polyps in about 85 per cent of cases, as well as melena, anemia, and fatigue. Abdominal surgery has been recommended only for obstruction or bleeding and not as a prophylactic measure. Radical or extensive surgical procedures have not been considered advisable.

These cases are unusual in that the typical pigmentation was absent in the father, despite the presence of polyposis of the small bowel and colon. Typical pigmentation was present in the brother of the propositus, but was not associated with polyposis.

Polyps of the Peutz-Jeghers syndrome have been considered to be hamartomas by Morson. This view is supported by others, and by the authors. The microanatomy of these polyps strongly suggests a developmental origin: the principal malformation is a localized arborizing proliferation of the muscularis mucosae. The epithelial covering is essentially normal.

Three cases of the Peutz-Jeghers syndrome in the same family, each representing a different facet of the syndrome, are reviewed.—*Stephen N. Tager, M.D.*

DEBRAY, CH., HARDOUIN, J.-P., PIRONNEAU, A., TERRIS, G., and MARTIN, ÉT. Les lipomes du colon. (Lipomas of the colon.) *Arch. mal. app. digest.*, Sept., 1963, *52*, 883–908. (From: Hôpital Bichat, Paris, France.)

Lipomas of the colon are rare, yet among benign tumors of the large bowel, rank second in frequency after simple adenomas. They are encountered more often after the fifth decade.

Anatomically, the subserous lipoma is the rarest; the submucous lipoma is more frequent and is well encapsulated. It has no particular vascular pedicle, but, it could be pedunculated or sessile. The mucosal covering could be ulcerated due to ischemia.

The symptoms of lipomas of the large bowel consist of pain, constipation and hemorrhage. However, clinically most of the lipomas of the colon are silent or asymptomatic and are discovered roentgenologically or during surgery. The larger lipomas are more prone to necrosis; hence, extrusion and healing.

The major complication of this tumor is an invagination of the large bowel. The roentgen signs of the colonic lipoma are those of a benign tumor: location in the proximal half of the large bowel; smooth borders; large volume; and increased translucency. The authors present 2 cases with several excellent roentgenograms.—*Jirair N. Sarian, M.D.*

WYCHULIS, ADAM R., JACKMAN, RAYMOND J., and MAYO, CHARLES W. Submucous lipomas of the colon and rectum. *Surg., Gynec. & Obst.*, Feb., 1964, *118*, 337–340. (From: Sections of Proctology and Surgery, Mayo Clinic and Mayo Foundation, Rochester, Minn.)

Between 1948 and 1962, 67 cases of submucous lipoma of the colon were found surgically at the Mayo Clinic. In 38 cases the operation was performed primarily for the lipoma, while in 29 the lesion was found incidentally.

The chief symptom was pain, sometimes colicky. Although blood was present in the stool almost one-third of the time, it did not correlate with ulceration of the tumor and was apparently due to other lesions. The tumors were rather uniformly scattered throughout the colon and were found in the cancer age group.

The preoperative diagnosis was made roentgenographically based on an intramural filling defect on barium enema study in 34 patients, and on the basis of radiolucency, smooth surface and changing shape on compression in 4 patients. Eight patients had intussusception. Four were found on proctoscopic examination.—*H. J. Klos, M.D.*

SANEN, FRANK J., MYERSON, RALPH M., and TEPLICK, J. GEORGE. Etiology of serious reactions to oral cholecystography. *A.M.A. Arch. Int. Med.*, Feb., 1964, *113*, 241–247. (Address: Dr. Myerson, Medical Service, Veterans Administration Hospital, University and Woodlawn Avenues, Philadelphia 4, Pa.)

The authors studied the effects of bunamiodyl (orabilex), sodium ipodate (sodium oragrafin), sodium tyropanoate (bilopaque) and iopanoic acid (telepaque) by administering the cholecystographic media to patients with portal cirrhosis and to patients with no evidence of either renal or hepatic disease. In addition, orabilex was administered in 9 gm. doses to 18 patients with chronic renal insufficiency.

The following observations were made: (a) orabilex did not aggravate the existing renal disease in those patients with renal insufficiency; (b) in the presence of marked hepatic disease, with or without jaundice, an increase in dosage of the cholecystographic medium will not force visualization of the gallbladder or biliary tract; (c) in the presence of hepatic disease, the opaque medium is shunted to the kidney for elimination and the resulting high blood concentration of the opaque medium may produce a systemic hypotensive defect; (d) in a considerable percentage of patients with severe hepatic disease, renal function, as measured by blood urea nitrogen, blood creatinine and creatinine clearance, was impaired following the administration of the cholecystographic medium; and (e) all normal patients had gallbladder visualization.

Renal failure following the administration of oral cholecystographic media is probably on the basis of several factors, included among which are: (1) the choice and dosage of the contrast medium employed; (2) the presence of associated parenchymal hepatic disease; and (3) hypotension.

The authors hypothecate that there is no basis in fact to the concept that a large dose of contrast medium will force visualization of the gallbladder in the face of severe hepatic derangement or biliary tract disease. Furthermore, increasing the dose may lead to rapidly developing high blood levels of the contrast medium, associated with which may be the development of significant systemic and/or renal hypotension. It is felt that the latter, resulting in a reduction of the glomerular filtration, is the primary mechanism of renal failure rather than a nephrotoxic effect of the opaque substances. In this regard, it is felt that the absorption index of the contrast medium is significant, and that the greater the absorption index, the lower should be the administered dose.

The clinician is admonished to adhere closely to the recommended dose of the cholecystographic medium that is employed, since the dosage is dependent upon the absorption index and elimination ratio, each of which varies with the different contrast agents. The administration of these compounds to patients suffering from coronary insufficiency should be carried out with caution and the observation of

blood pressure readings is recommended.—*John H. Harris, M.D.*

Genitourinary System

Ciampelli, L. Su due casi di sclerolipomatosi perirenale con spostamento anteriore della pelvi ed uretere associata a calcolosi ureterale. (On two cases of perirenal sclerolipomatosis with anterior displacement of the pelvis and ureter associated with ureteral calculi.) *Radiol. med.*, Nov., 1963, *49*, 988–1002. (Address: Badia Prataglia, Arezzo, Italy.)

Two cases of perirenal sclerolipomatosis are reported.

In the first case, a male of 52 years of age, the intravenous urography demonstrated a calculus in the lower third of the left ureter and an incomplete visualization of the pelvis of the left kidney. Retrograde pyelography revealed an irregular filling defect of the pelvis of the left kidney and a minimal but definite anterior displacement of the pelvis and of the upper third of the ureter. These findings were thought to be the expression of a retroperitoneal neoplastic mass and the patient was operated on. At surgery, the pathologic finding was found to be due to perirenal sclerolipomatosis.

In the second case, a male of 48 years of age, intravenous urography showed calcifications in the left kidney, a radiopaque calculus in the pelvis, and incomplete opacification of the calyceal system. Retrograde pyelography demonstrated also an anterior displacement of the pelvis and of the proximal third of the ureter. A diagnosis of renal calculi, associated with displacement of the pelvis and upper third of the ureter due to perirenal sclerolipomatosis, was made. The diagnosis was confirmed at surgery.

The author believes that perirenal sclerolipomatosis is a peripyelitic reactive phenomenon similar to the inflammatory reactions noted in chronic inflammatory processes. This hypothesis is based on the relative frequency at surgery of a thickening of the fatty tissue in the perirenal area in patients with a history of chronic kidney disease. In the 2 cases presented, the sclerolipomatous reaction was thought to be in relationship with the long standing effect of the calculi, even though in the first case it was not in the kidney itself.

To explain the anterior displacement of the pelvis one has to remember that the kidney is lodged between the two sheets of the renal fascia; one, anterior, rather thin; and one, posterior, more resistant, which is adherent to the fascia of the quadratus lumborum. An increase in the content of the renal fascia, as might happen in an inflammatory process, will produce an increased pressure on its walls. The anterior wall, being less resistant, will tend to be displaced anteriorly. Also to be considered is that the tissue most likely to be involved in the perirenal inflammatory process is localized especially in the anterior portion of the renal lodge and around the hilus of the kidney; therefore, a hyperplastic reaction in this tissue will displace anteriorly the pelvis and part of the upper third of the ureter.

The possibility of perirenal sclerolipomatosis should be kept in mind in the differential roentgenologic diagnosis of expansive retroperitoneal processes which can be demonstrated indirectly by displacement of the pelvis of the kidney and of the ureter. The main points in the differential diagnosis are: 1) anterior displacement of the pelvis and of the most proximal segment of the ureter on an axis perpendicular to the frontal plane; 2) this anterior displacement is not associated with a vertical or horizontal rotation of the pelvis; 3) in the roentgenograms taken during intravenous urography, the pelvis and the ureter at the uretero-pelvic junction demonstrate an incomplete and inhomogeneous opacification; and 4) the posterior face of the pelvis and ureter, as visualized by the contrast medium, presents a semilunar contour, concave posteriorly.—*A. F. Govoni, M.D.*

Brannan, William, Birchall, Robert, Batson, Hugh M., and Kittredge, W. E. Dehydrated-hydrated excretory urography in the diagnosis of renal arterial hypertension. *J. Urol.*, Jan., 1964, *91*, 26–32. (From: Departments of Urology and Internal Medicine, Ochsner Clinic and the Department of Research, Alton Ochsner Medical Foundation, New Orleans, La.)

Numerous observers have utilized the principle that an ischemic kidney has a reduced blood flow and accordingly a lower rate of urinary flow resulting in increased concentration of para-amino-hippurate, inulin, and creatinine and decreased excretion of filtered sodium and water. These facts suggested to the authors that almost as much information might be obtained by a dehydrated-hydrated excretory urographic study as by a differential renal study. In the truly dehydrated patient, one can predict that the concentration of the opaque medium on the affected side will be equal to or slightly less than on the unaffected side. When the patient is later hydrated, and is undergoing maximum water diuresis, the concentration in the pelvis of the affected kidney will be inevitably greater than that in the pelvis of the unaffected kidney. Thus, hydration will "washout" the picture of the pelvis of the normal kidney and leave intact that of the affected kidney.

The authors prepare their patients with castor oil and then the patients are dehydrated overnight. The excretory urogram is obtained after rapid intravenous injection of 30 cc. of a 50 per cent hypaque solution through a 16 gauge needle. Fifteen seconds after completion of the injection a film is exposed and then

additional exposures are made at 2, 3, 4, 5, 10, and 15 minutes after the injection. If there is bilateral renal function, the patient is prepared for the hydrated study the next day. Nonfunction on either side precludes further study. In order to hydrate the patient, partaking of fluids is encouraged until 1 hour before the study when 1 liter of tap water is given within 15 to 20 minutes. He then drinks 8 ounces of water every 15 minutes until the test is terminated. After the bladder is completely emptied, 30 cc. of 50 per cent hypaque is injected intravenously and films are exposed at 5, 10, and 15 minutes after the injection.

When the patient is dehydrated, the ischemic kidney will ideally show significant decrease in size as compared to its mate, a delayed nephrogram, prolongation of the nephrographic phase, and delay in calyceal opacification if enough function remains to concentrate the opaque medium. Following hydration, the ischemic kidney will reabsorb water excessively and concentrate the opaque medium so that a "normal" pyelogram will be evident. In the opposite kidney (the functionally normal kidney) the opaque medium is so diluted that only a faint trace is seen. Interpretation depends on the distinct difference in the opacification in the collecting system, and any other minor differences are not considered abnormal. No abdominal compression is to be used during either the hydrated or the dehydrated phases of the study.

The authors concluded that the nephrographic phase was occasionally difficult to assess, but they did note that the nephrogram of the involved kidney in all patients either appeared later or persisted longer than it did in the normal kidney. Assessment of the concentration of opaque medium in the two kidneys was felt to be difficult and somewhat unreliable with the delay in appearance time of the medium in the calyces being more reliable. When the authors recorded separately the renal size, the nephrogram, the appearance time, the calyceal opacification, the concentration of the opaque medium in the dehydrated-hydrated state, and the occasional apparent absence of function on one side, they encountered no false-negative urograms. They note that the technique may not prove valid for a segmental lesion in one kidney or if there is bilateral disease in which the degree of stenosis is about the same. Significant elevation of the blood urea nitrogen level, with associated renal insufficiency, may invalidate the test.—*George W. Chamberlin, M.D.*

Shenkin, Henry A., Pollack, Howard, Somach, Fredric, and Bijaisoradat, Surin. Value of routine urography during cerebral angiography. *J.A.M.A.*, Jan. 18, 1964, *187*, 207–211. (Address: Dr. Shenkin, Episcopal Hospital, Philadelphia, Pa. 19125.)

In view of the fact that there is not infrequent association between renal and cerebral disease states, it has been the custom for some time on the neurosurgical service at the Episcopal Hospital, Philadelphia, Pennsylvania, to obtain a routine study of the urinary tract in every patient undergoing cerebral angiography. The roentgenogram of the abdomen is taken 5 minutes after the injection of contrast material for demonstration of the cerebral vessels, a timing which has proved more satisfactory than the 10–15 minute interval initially used. In a series of 275 patients so studied, 24 (9 per cent) showed clinically significant and generally unsuspected abnormalities of the genitourinary tract. Common abnormalities previously known or available for diagnosis on physical examination were not included in the 24. In another 11 patients (4 per cent) abnormalities of interest, if only of relatively minor significance, were detected.

In 2 of 3 patients with cerebral aneurysms, polycystic renal disease was demonstrated. Ten patients were found with unilateral renal enlargements of which 4 were associated with neoplastic cerebral disease. Of these 2 proved to be metastatic carcinoma to the brain with the kidney likely as the primary site of tumor growth, and 1 was due to metastasis of a carcinoma of the breast that had also spread to the brain. An atrophic kidney was detected in 4 patients, 3 with associated occlusive vascular disease, 2 of whom were hypertensive.

In the absence of urologic symptoms, the roentgenogram of the abdomen following cerebral angiography, taken for demonstration of the urinary tract, appears to be a worthwhile addition to the procedure in that it will reveal disease that might otherwise go undetected.—*Lois Cowan Collins, M.D.*

Simmons, John L., and Jones, M. A., Jr. Use of the renal scintiscan in urology. *J. Urol.*, Nov., 1963, *90*, 642–654. (From: Department of Surgery [Urology] and the Department of Radiology, University of North Carolina, Chapel Hill, N. C.)

This report is a review of 166 scintiscans with the patient's history, physical examination, blood urea nitrogen level, excretory urogram and scintiscan all being reviewed to learn what relation there might be between various renal diseases and the appearance of the excretory urogram and the scintiscan. It is an attempt to note conditions which alter the scintiscan and to determine in what situations the scan may contribute some additional information for the evaluation of various renal abnormalities.

Mercury 203 labelled chlormerodrin was used in preference to I^{131}. The labelled chlormerodrin has been found to be primarily excreted by the renal tubules, with glomerular filtration accounting for only about 1.5 per cent of the excretion of mercury in

the urine. A sufficient amount of mercury, however, is retained in the renal tubule cell to enable the renal outlines to be traced by an external counting device for a period of over 24 hours. The authors employed a Picker magnascanner with a 19 hole collimator, focused at $2\frac{3}{4}$ inches and a 50 per cent isoresponsive line width of about $\frac{5}{8}$ of an inch. The scanning procedure was performed in the 2 to 6-hour period following the injection of 75 μc of mercury tagged chlormerodrin. The procedure was used to evaluate patients with renal hypertension, uremia, renal tumors, obstructive uropathy, and post-traumatic renal damage.

The results obtained from the study of patients with known renal hypertension have shown a good correlation between the excretory urogram and the renal scan in determining the size of the functioning renal mass. However, the scan was found to be inconsistent in many instances when the alteration of the concentration of the mercury scan might be expected to give additional information. The scan was not found to be a satisfactory screening test for hypertension secondary to renal artery stenosis and should be used as a supplementary study, rather than as a substitute, to pyelography or aortography techniques.

As has been demonstrated with the excretory urogram, an increase in renal insufficiency (as in uremia) results in a decrease in Hg^{203} concentration until finally no renal pattern is apparent. Ultimately, the scan will only show hepatic outline. It has been found that with blood urea nitrogen concentrations up to the 30 to 40 mg. per cent range there is fairly good correlation between the appearance of the scan and the excretory pyelogram. With increase in renal insufficiency there are occasions when an excretory urogram will show nonvisualization, whereas the scan will continue to show a good renal outline.

The renal scan has only occasionally been of additional aid, over that provided by pyelography, in discovering renal tumors. Large lesions in the renal parenchyma will be displayed on a renal scan. However, in the detection of peripelvic cysts of 3 cm. or less the scintiscan may appear normal. In fact, it is probable that any mass deeply situated in the kidney of 3 or 4 cm. size or less may be missed not only because of its size but also because it may be surrounded by sufficient normally functioning parenchyma to "mask" its location.

In cases with complete obstruction of the ureter, it has been discovered that the excretory urogram will show nonvisualization at a time when the scintiscan would still show functioning renal tissue. It is suggested by the authors that the persistence of renal function by scintiscan may be a guide in determining whether or not the involved kidney will regain function after the obstructive uropathy is relieved.

The scintiscan has been found to be more reliable in cases of renal trauma than is the excretory pyelogram.

One may conclude from this article that the renal scintiscan does not replace either excretory urography or pyelography in the evaluation of renal disorders. It is a useful adjunct to the radiologist's armamentarium and in selective cases may provide the additional information necessary to make a definitive diagnosis.

This article includes 20 excellent figures.—*George W. Chamberlin, M.D.*

WINTER, CHESTER C. Radioisotope uroflometry and bladder residual test. *J. Urol.*, Jan., 1964, *91*, 103–106. (From: Division of Urology, Department of Surgery, Ohio State University Hospital, Columbus, Ohio.)

Information concerning the patient's ability to urinate and evacuate the bladder is frequently desirable. When the radioisotope method of uroflometry is used, a scintillation counter with rate meter and recorder constitutes the necessary equipment. An average dose of 5 μc of radiohippuran is recommended. A measurable level of radiation is achieved in the bladder in 30 minutes after intravenous administration, and in 60 to 90 minutes after the oral administration. The patient's bladder should be nearly full without overdistention at the time of the test. The scintillation counter is placed against the posterior aspect of the bladder and a record of the level of radioactivity is made. The paper speed is then increased to 6 inches per minute and the patient instructed to void.

As the bladder empties, the level of radioactivity diminishes and is recorded as a descending curve in the tracing. The maximal rate of descent of the tracing reflects the patient's optimal flow rate and together with the consistency of the pattern is taken as an index of micturition ability. The degree of emptying is shown by the amount of residual bladder radioactivity. Division of the initial peak of vesical radiation into the residual bladder radiation level gives the percentage of residual urine in the bladder, using the background count as a baseline. The recorded data may also be transformed into mean flow rates of milliliters per second, since the measured voided specimen is available.

The author concludes that the radioisotope uroflometry technique correlates well with catheterization residuals, urethrocystoscopy, urethrography, cystography and cystometry. The advantages of the method include: simplicity, permanence of the record, accumulation of dual data on micturition ability and bladder residual, possibility of serial studies (such as pre- and postoperative evaluations) and last but not least no hazard or trauma to the patient. The disadvantages include the necessity of possessing radiation detecting equipment and the realization that false positive results can occur if the patient is not at ease or is uncooperative.—*George W. Chamberlin, M.D.*

MASLOFF, JAMES I., and BUNTS, R. CARL. The carbon dioxide cystogram. *J. Urol.*, Nov., 1963, *90*, 658–664. (From: Urology Section, Surgical Service, McGuire Veterans Administration Hospital, Richmond, Va.)

The authors acknowledge that persistent vesicoureteral reflux is a significant pathologic entity particularly in patients with a neurogenic bladder. Severe reactions to cystography have been of frequent occurrence in their patients most of whom have had spinal cord injury. Because of this, carbon dioxide cystography was performed in an effort to reduce the morbidity attending the ordinary cystography. A total of 69 patients, 63 with spinal cord disease and 6 without neurogenic bladders, were evaluated. Seventy-six CO_2 cystographies and 57 contrast medium cystographies were done with the gas cystography one day and the contrast medium cystography the following day. Carbon dioxide was chosen because of its wide margin of safety as indicated by high solubility in plasma at 37° C.

In performing the contrast medium cystography, a Foley catheter was inserted under aseptic conditions and the bladder irrigated with a clorpactin solution. Contrast medium was instilled into the bladder at the rate of 30 cc. per minute by gravity drip method, usually 100 cc. of medium being employed in a quadriplegic and 150 cc. in a paraplegic patient. After this a Cunningham clamp was placed around the penis, the catheter was clamped, and the initial film was exposed. When possible a 20-minute delayed cystogram was obtained.

The carbon dioxide cystography was performed using 100 per cent carbon dioxide with the gas being delivered in a measured volume with the aid of a 50 cc. Luer Loc syringe and a 3 way stopcock. Insertion of the catheter and bladder irrigations were effected as described for the contrast medium cystography. CO_2 (150 cc. 100 per cent) was injected into the bladder at the rate of approximately 100 cc. per minute. If the patient could tolerate the procedure, the catheter was momentarily opened and another 150 cc. CO_2 was injected. Cystometric determinations were made by connecting the Foley catheter (by means of a needle and a 20 inch segment of intravenous tubing) to a mercury manometer.

Fifty-seven paired cystograms were available for comparison. Reflux was demonstrated in 16 pairs. Of these 16, identical correlation as to side and degree of reflux was demonstrated in 4. In one other case the correlation was essentially identical in that reflux appeared on the same side on both studies, but differed in degree on one side. Thus in 5 of the 16 cases, there was essentially complete correlation between the contrast medium cystogram and the carbon dioxide cystogram. Therefore, there were 11 cases in whom reflux had been demonstrated, but in whom complete correlation was absent. When all patients are included, essentially complete correlation was found in 81 per cent of the 57 paired cystograms.

No real difference as regards the reaction to the two types of cystographies could be determined.

The authors conclude that the carbon dioxide cystogram may be of value as a procedure for use especially in the patient with reflux who has had reactions to previous contrast medium cystography and in whom follow-up studies are necessary. They do state, however, that the CO_2 cystogram is not reliable for detecting fine shades of incomplete reflux.

This article includes two tables and three figures.—*George W. Chamberlin, M.D.*

McGOWAN, LARRY. Diverticula of the urethra in women. *G. P.*, Feb., 1964, *29*, 88–91. (From: Department of Obstetrics and Gynecology, Stritch School of Medicine, Loyola University, Chicago, Ill.)

Confirmation of the clinical impression of urethral diverticula in women may be easily made roentgenographically by employing contrast medium. Fifty-seven female patients, 29 of whom had dysuria ranging from a few months to 5 years as chief complaint, are presented. Most had panendoscopic and roentgen examinations. Aside from direct instillation of the opaque medium via a needle or urethral catheter, or fortuitous demonstration of the urethral pouch during excretory urography or voiding cystography the largest group (24) was investigated by inserting a urethral catheter with the openings at the tip occluded. The filled retention bag was pulled securely against the vesicourethral junction and, while occluding the meatus, the medium was allowed to fill the urethra through the holes previously made in the catheter proximal to the retention bag. Films were exposed immediately. If possible, previous emptying of the diverticulum by digital pressure was done.

The panendoscope was the most efficient in the opinion of the author for identifying the orifice or orifices of the sac. Urethrograms as described defined the size, shape and location.

Surgical removal is recommended and described.—*Newell Richardson, M.D.*

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Case Two: Severe aortic stenosis with calcified aortic valve cusps

A 63-year-old woman with a clinical picture of tight aortic stenosis had been ill with precordial pain on exertion. Chest radiographs indicated minimal cardiac enlargement (Figure 1). Serial percutaneous transfemoral retrograde aortograms showed severe aortic stenosis (Figure 2). In other aortograms, calcification of cusps was noted. There was no

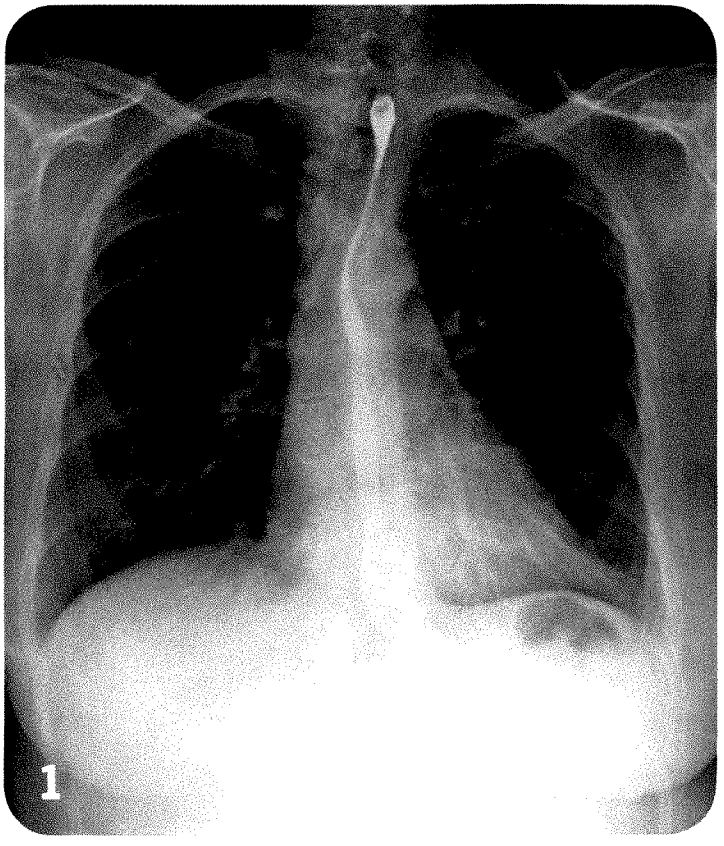


Figure 1: Preoperative radiograph. Note minimal cardiac enlargement.

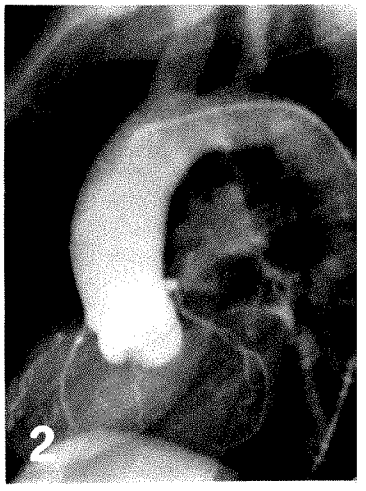
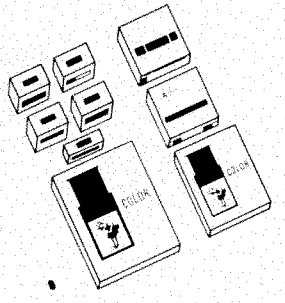


Figure 2: Retrograde aortogram shows severe aortic stenosis.



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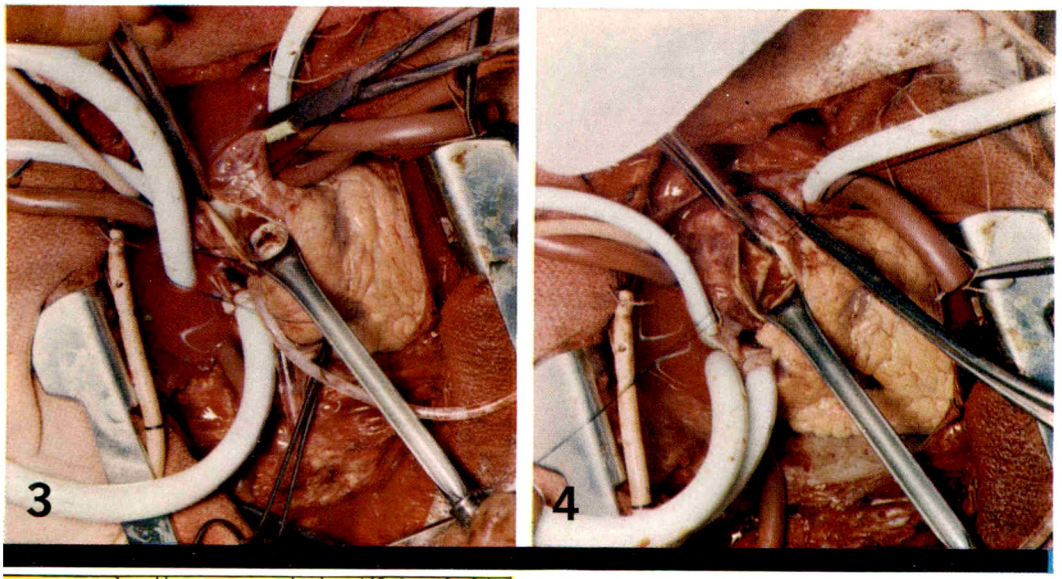
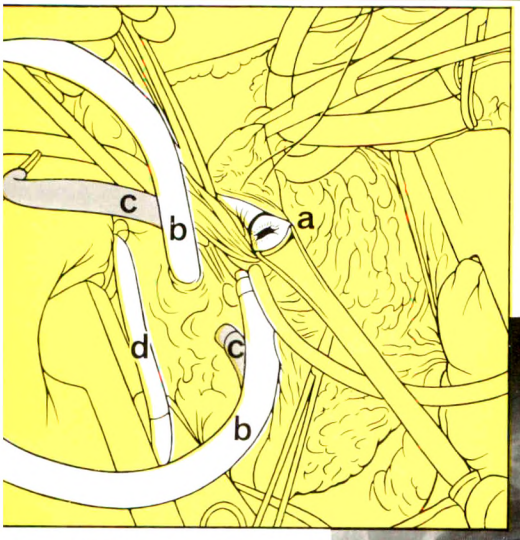


Figure 3: Aortic valve prior to valvulotomy.

Figure 4: Aortic valve following valvulotomy.



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. Stenosed aortic valve
. Drainage tubes in venae cavae
. Snares (occluding tapes) about venae cavae
. CO_2 catheter

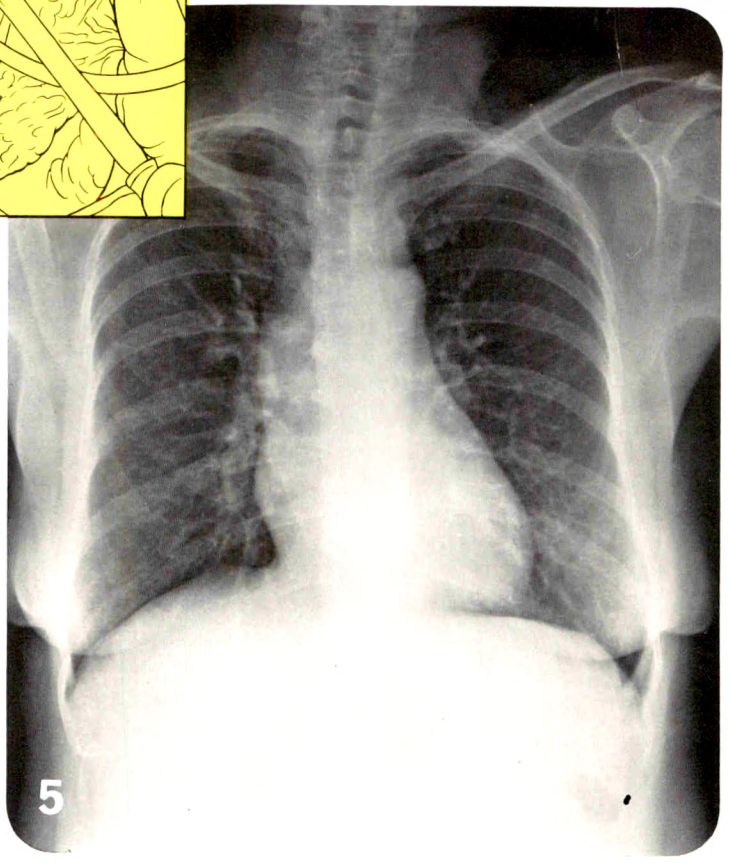
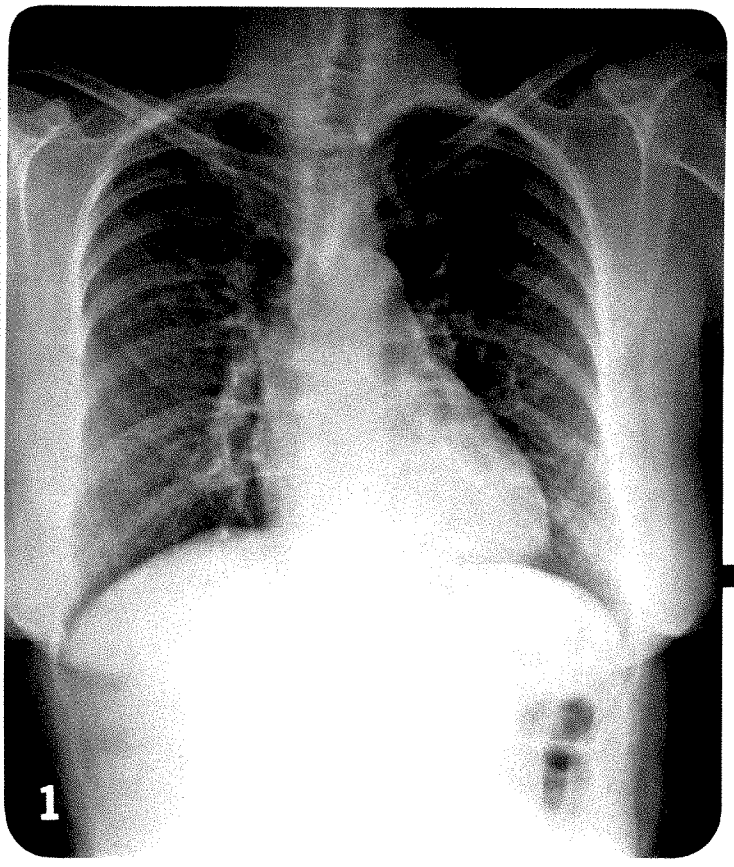


Figure 5: Postoperative radiograph shows decrease in heart size.

For details of second case—turn to pages 3 and 4.

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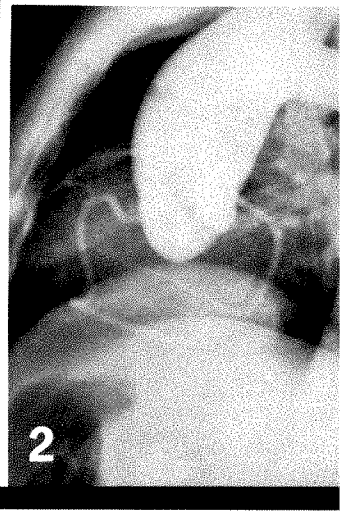


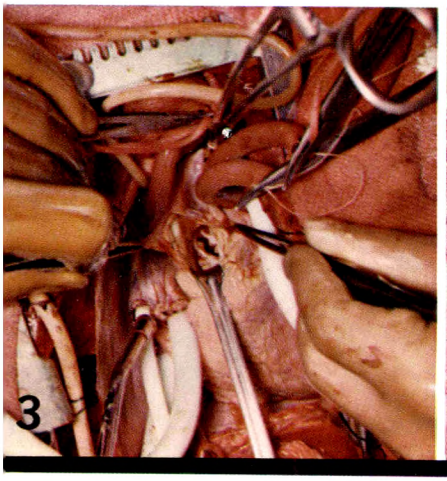
Figure 1: Preoperative radiograph of chest.

Figure 2: Preoperative aortogram.

Acquired Heart Disease: Two cases

Case One: Aortic stenosis with six months' history of precordial pain and dizziness

A 59-year-old woman with physical signs and symptoms of aortic stenosis gave a six months' history of precordial pain and dizziness. Preoperative radiographs of chest showed moderate cardiac enlargement (Figure 1). Serial percutaneous transfemoral aortograms revealed a markedly dilated ascending aorta, and an aortic valve that was stenotic and deformed with calcifications (Figure 2). There was no aortic insufficiency. The coronary arteries appeared normal. At surgery, a tightly stenosed aortic valve was found (Figure 3). Calcium was removed and the leaflets were mobilized (Figure 4). Postoperative course was uncomplicated and patient improved clinically. Postoperative radiograph indicated decrease in size of heart (Figure 5).



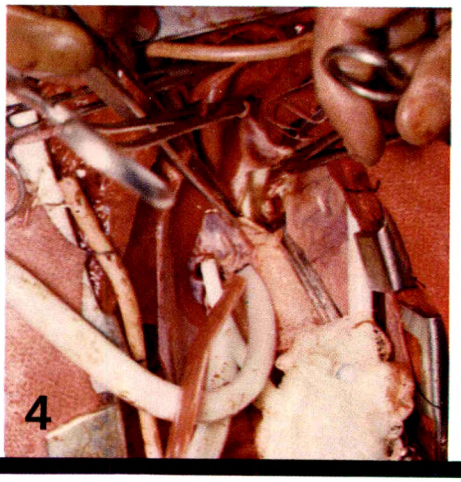


Figure 3: Aortic valve before valvulotomy.

Figure 4: Aortic valve following partial removal of calcium.

indication of aortic insufficiency. However, there was marked poststenotic dilatation. Coronary arteries were normal.

At surgery, a tightly stenotic, heavily calcified aortic valve was found (Figure 3). Calcium was removed and the leaflets mobilized (Figure 4).

Patient was greatly improved following operation. Postoperative radiograph showed a decrease in heart size (Figure 5).

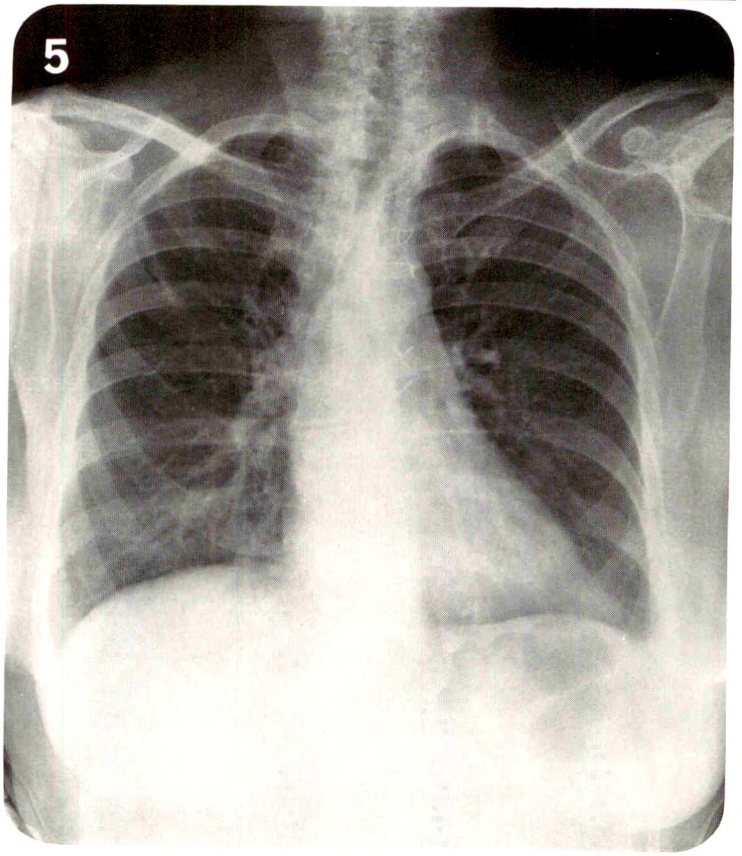


Figure 5: Postoperative radiograph. Note decrease in heart size.

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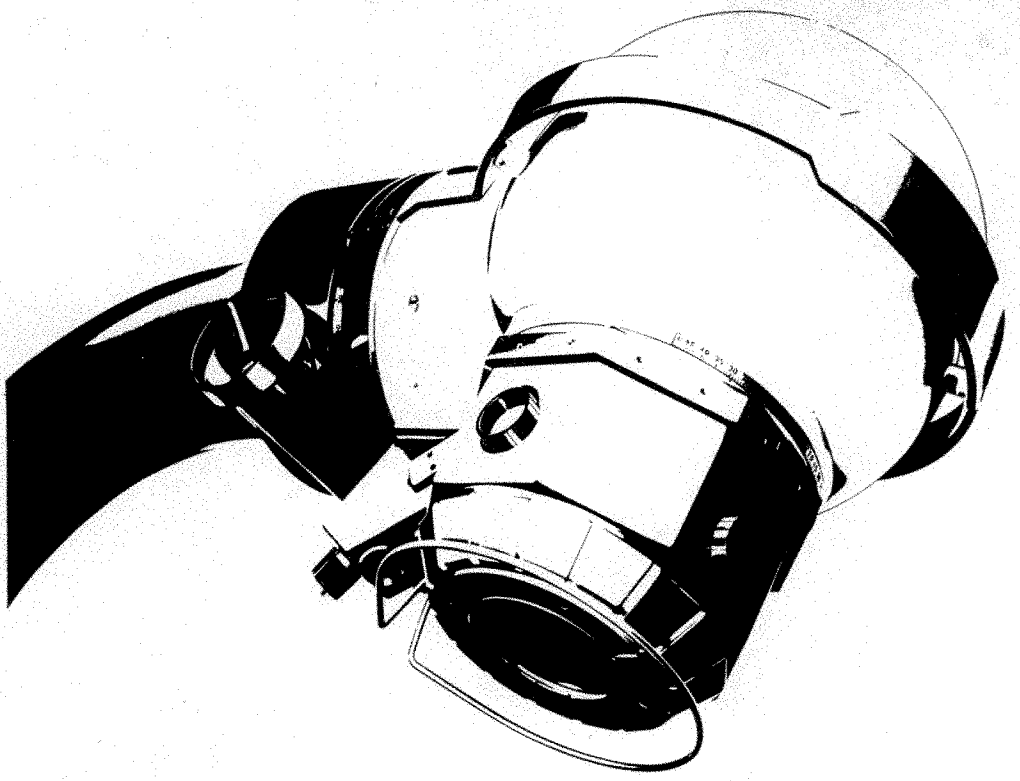
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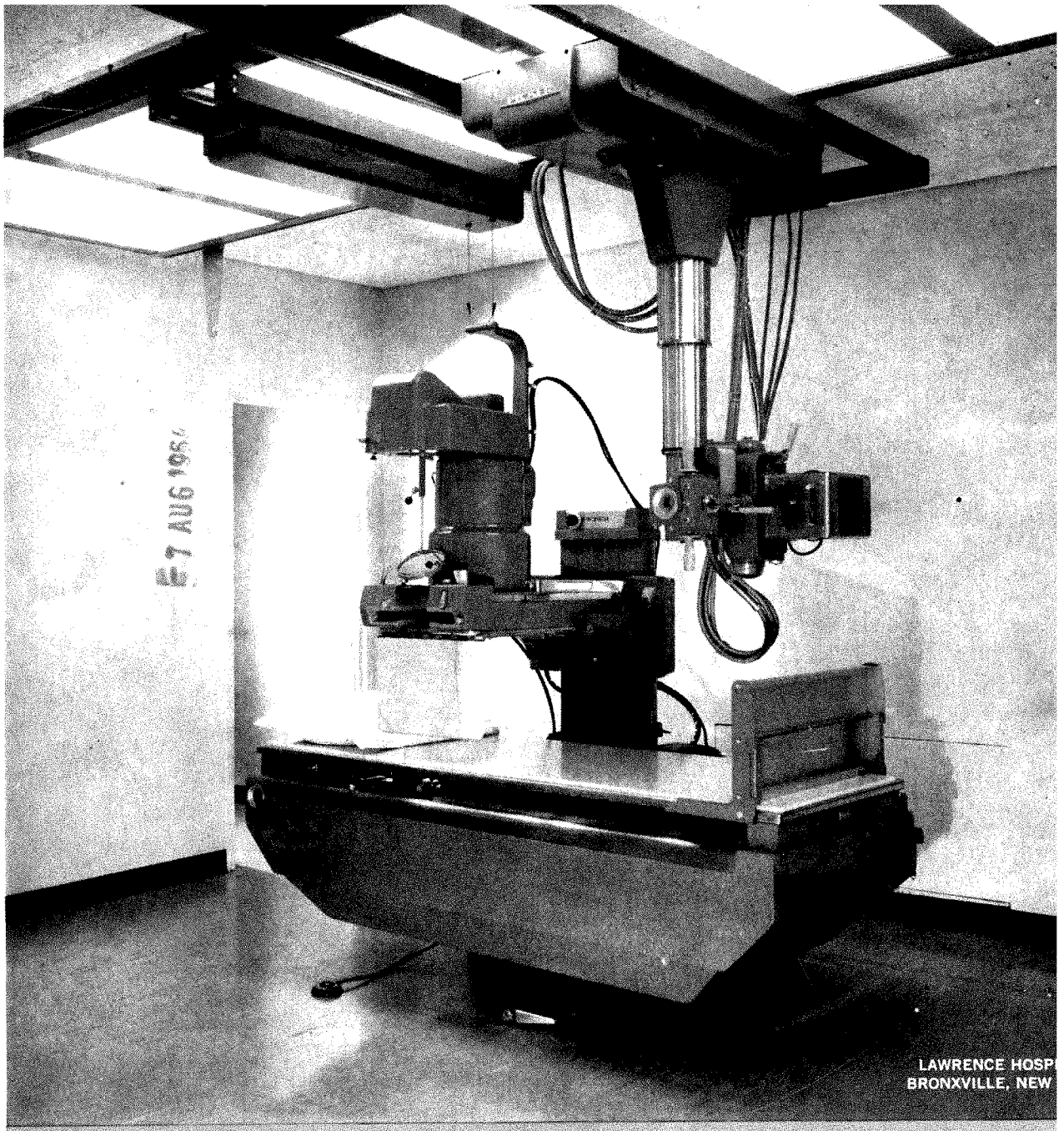
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